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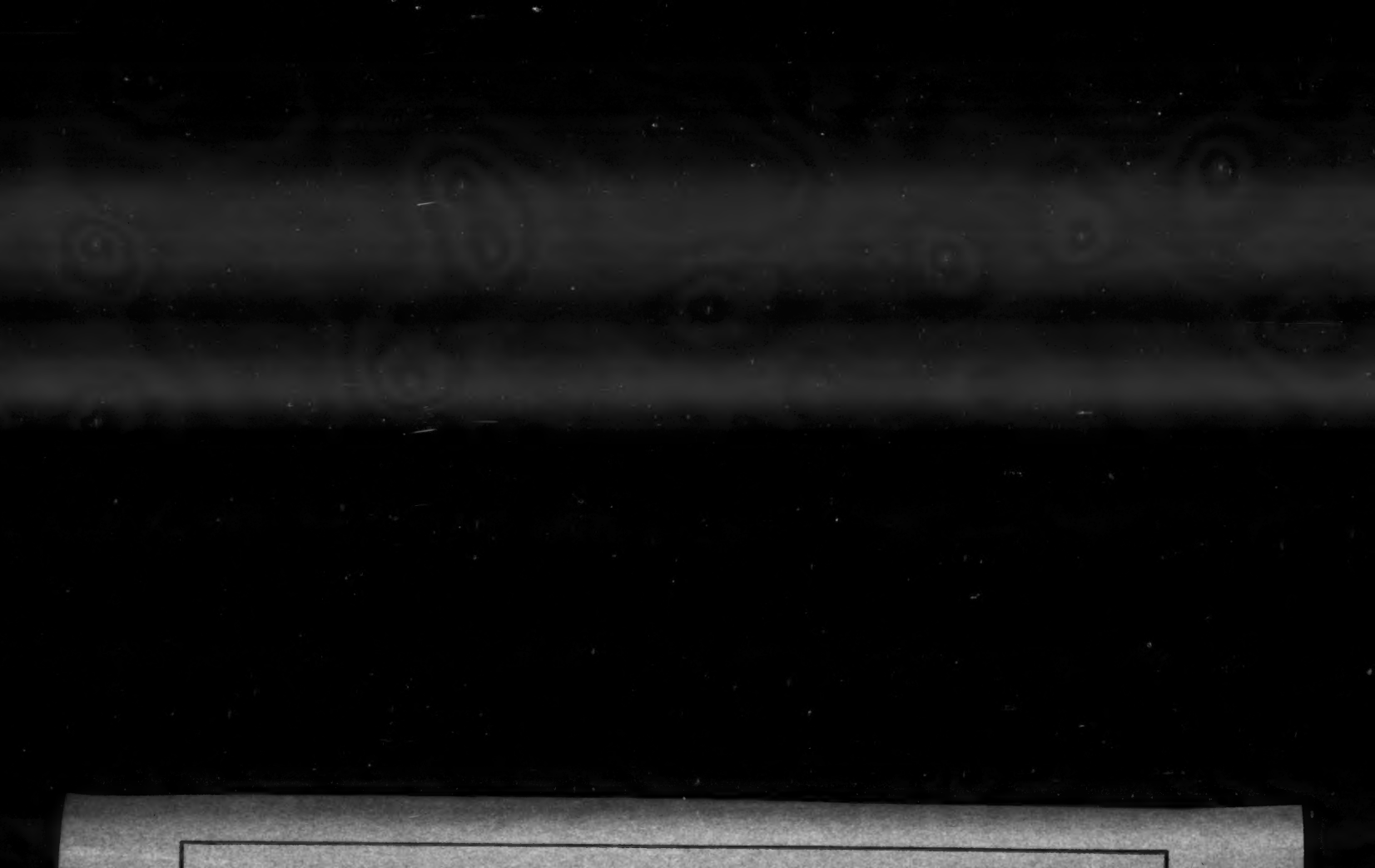

Lahey Clinic Boston Number

THE SURGICAL CLINICS
OF
NORTH AMERICA

Volume 4 **Number 6**

THE EVOLUTION OF A THYROID CLINIC

FRANK H. LAHEY

IN our clinic at the New England Deaconess and New England Baptist Hospitals we have, up to the present, passed through three distinct phases in the treatment of toxic goiter. When we first undertook the management of toxic goiter the mortality of surgery in this disease was so appalling that it was but natural that improvement in this depressing situation should become the primary and inspiring task imposed upon us.

In surveying the situation as it then existed it became at once apparent that thyroid surgery was of such a nature that it could not be conducted under the same plan as could the surgery of appendicitis, gall-stones, gynecologic procedures, etc. It was evident to us, for example, that we could not go to a suburban hospital, see there a case of exophthalmic goiter (primary hyperthyroidism), operate upon it casually, leave it to the family physician to care for, and expect to maintain a mortality rate superior to that already existing at that time.

We therefore insisted, since toxic goiter was of such seriousness and so prone to assume sudden alarming and unusual phases, that its successful management demanded for the cases centralization, individualization, and specialization.

We maintained that all goiters should come to our hospitals, where we were particularly equipped for the care and management of these cases; that they be anesthetized only by our own anesthetist, trained and experienced in the adminis-

tration of gas oxygen to these cases, trained and experienced in the management of the urgent conditions which occasionally arise in the surgery of toxic goiter; that they be operated by a non-rotating group as far as the important members of the operating team went, and that their after-care be in the hands of this same group plus a group of nurses trained and experienced in the postoperative handling and management of goiter surgically treated, a factor which I assure you has played no small part in maintaining the low mortality rate which we have been able to attain.

We next learned that it was possible, by means of this organized group, to be almost absolutely certain that such a thing as a table death would not occur, because of our experience in selecting cases for operation and because of the experience of our anesthetist in detecting possible surgical calamities on the table sufficiently early to so limit us to the degree that this did not occur.

Subsequently, however, we were confronted with a factor much less tangible, and one which played then, as it does now, by far the most important rôle in the production of mortality in the surgical treatment of toxic goiter: that is, the unknown degree of postoperative thyroid reaction which occurs on the day of operation or the day following it. We found that a certain number of toxic cases endured the complete procedure of subtotal thyroidectomy on both lobes, left the table in such a condition as to cause us no alarm, and within twenty-four hours passed into a stage of such extreme activation with delirium that, in spite of all measures to combat it, they terminated fatally within a day or two after the operation.

We further found that not only was our mortality augmented by these untoward postoperative reactions but also by the fact that we had at times allowed ourselves to become deeply involved in an extensive operation that did not readily permit of change to a more limited procedure on a patient, seemingly in good condition, only to find too late such a rapid change in the patient's condition that a mortality usually occurred on the same day.

This, then, led us to what we have called multiple stage procedures, the plan which we now pursue.

The surgical procedure in toxic goiter permits of division into steps, each a block, so to speak, which may be considered a measure of progress, each to be completed in the doubtful cases as a single procedure. The next step is undertaken only after the effect of the first has been noted and the probable outcome of an advance through the next one seriously considered, with the distinct conviction that where doubt exists as to the outcome, only the first step shall be used. The patient is returned to bed, the effect noted, and, if the reaction be not unduly severe, the next step completed within two or three days.

The various steps, or blocks, then, of these procedures are ligation of the right superior pole, ligation of the left superior pole, ligation of the right inferior thyroid artery, ligation of the left inferior thyroid artery, elevation of the skin-flap, right subtotal hemithyroidectomy, and left subtotal hemithyroidectomy. The application of these steps to a given case will be taken up after the consideration of the next phase through which we have passed.

In our early experience with toxic goiter, particularly of the exophthalmic or primary hyperthyroidism type, we were occasionally confronted with a type of patient who had tachycardia, nervousness, tremor, and occasionally an enlargement of the thyroid, who stated that she became easily exhausted, but who usually had lost no weight, who had neither the eye signs of exophthalmic goiter nor the inconstant secondary features of the disease, such as moist skin, diarrhea, cessation of menstruation, itching, hyperalertness, etc. Operation unwisely performed upon 2 or 3 patients of this type failed to yield the gratifying results which are so constant in the properly conducted surgery of toxic goiter. We very soon recognized that there existed a group of patients with symptoms closely simulating those of exophthalmic goiter or primary hyperthyroidism, but quite evidently, from our experience with them, not due to thyroid activity, and not benefited by procedures directed at that structure.

At that time there developed two factors destined to provide an extensive experience with cases of this kind. On the one hand, the war, with its large group of N. C. A. cases, or D. A. H. cases, as they are called by the English, and called by army surgeons "soldier's heart." On the other hand, the basal metabolism test, a measure by means of which it is possible to distinguish, almost with certainty, these N. C. A. cases with tachycardia of non-thyroid origin from those of thyroid origin.

It was our lot during the war to have the opportunity of observing a considerable number of these cases, and it was the good fortune of Dr. B. E. Hamilton, the cardiologist to our clinic, to be assigned under Sir Thomas Lewis in England, when that astute clinical observer was engaged in the close study of this trying group of cases, a large number of which developed in the armies of all countries. It was furthermore our good fortune, following the war, to have referred to us by the Army Department a considerable number of cases of this type which, under the mistaken diagnosis of hyperthyroidism, had been unsuccessfully treated in army hospitals by various measures, such as prolonged rest, *x*-ray, and even by surgery. So it was but natural, again, that the next phase through which we passed should be the development of measures for the critical study of these cases suspected of toxicity, in order that all cases of tachycardia and neuroses of non-thyroid origin be eliminated, and cases only of proved thyroid toxicity be operated upon.

Following a considerable experience with basal metabolism tests,[1] we have observed with reliable constancy the following facts: that active hyperthyroidism does not exist in the absence of an elevated metabolism, that following subtotal thyroidectomy the patients are not cured of hyperthyroidism unless their basal metabolism comes to and remains at normal, that if the rate does not come to normal, it may usually be brought to normal and the persisting signs of hyperthyroidism eliminated by a further removal of a sufficient amount of thyroid tissue.

[1] Dr. S. M. Jordan, of this clinic, is now reporting a review of 5200 metabolisms on 1635 patients.

In reviewing our basal metabolism experience and our thyroid operations, 2373 (September 23, 1924) in number, 42.4 per cent. of which were exophthalmic or primary hyperthyroidism and 25.4 per cent. secondary hyperthyroidism or toxic adenomata, 67.8 per cent. were on toxic goiter, we were convinced that there are two causes of failure to cure hyperthyroidism by surgical measures—one was the factor just mentioned (the removal of an insufficient amount of thyroid tissue), and the other, the factor which concerns the N. C. A. group to which reference was made above, namely, that cases are operated upon who are not suffering from hyperthyroidism, but from the condition of N. C. A. which often so closely simulates it.

It is here, then, that critical basal metabolic studies must be made, since the basal metabolism readings in N. C. A. are of normal values, while those of hyperthyroidism are always elevated. In the basal metabolism studies of these cases it should be realized that N. C. A. cases are notably poor subjects for the basal metabolism test, inasmuch as they adjust themselves badly to the measures of the test because of their apprehension. It therefore becomes necessary that repeated tests, preferably each on succeeding days, be made, so that it may be ascertained whether or not the rate remains persistently high, as is the case with toxic goiter, or gradually progresses toward and reaches normal, as is the case with N. C. A., when the nervous individual becomes adjusted to the measures necessary to the conduct of the test.

Our present method of dealing with toxic goiter, then, is to send the cases to the hospital for critical general, cardiologic (to be spoken of as the third phase), and basal metabolic study, and having determined to our satisfaction the existence of hyperthyroidism, to submit them to surgery under the following plan: They are given scopolamin, gr. 1/200, and morphin, gr. $\frac{1}{4}$, two hours before operation, and provided there is no unusual reaction, such as marked respiratory depression or extraordinary pulse acceleration, the same dose, in an individual of average size, is repeated one hour before operation. The patient is then brought to the operating-room for operation, but the decision

as to the type and extent of operation is deferred until the patient is on the table, under gas, draped, and ready for operation. Then, based upon the clinical history, such as length of the disease, clinical evidences of its intensity, such as loss of weight and pulse-rate at the time of examination, the basal metabolic rate, the cardiologist's report as to cardiac crippling, and, finally, the anesthetist's report as to the patient's immediate condition, which is the condition under which he or she must endure the procedure and in most cases represents the condition at its worst, a decision is reached as to whether or not the patient will successfully stand an operation directed immediately toward removal of part or the whole of the thyroid gland. If it is evident from the factors above stated and the anesthetist's report of the immediate condition, based largely upon pulse-rate and pulse-pressure, that the patient's condition is so doubtful that the outcome of even a single ligation is uncertain, the patient is returned to bed following the administration of the gas oxygen only, and the reaction to this procedure is noted. If there is not an alarming reaction to this, we may be quite sure that the ligation of one superior pole will be endured, following which the patient is returned to bed for two or three days, and the other superior pole may then be ligated. We have had one death from the ligation of the first pole. We have never had a death from the ligation of the second pole, and we have had no other deaths from pole ligations. They are then sent home for six weeks, during which time they usually gain from 10 to 20 pounds in weight.

On returning to the hospital the situation is the same, then, in the patient who has had the poles ligated as in the case of the patient whose condition on first going to the operatiog room is such that it seems wise to make the first surgical procedure an operation directed toward a subtotal removal of one or both lobes of the thyroid, except that in the patient whose poles have been preliminarily ligated there is evidence as to how he stood the preliminary measures. This evidence is the anesthetist's five-minute interval record of the patient's pulse, respiration, and blood-pressure during the previous procedures

plus the degree of postoperative reaction which has been ascertained and noted by the anesthetist on his anesthesia chart.

In either case, then, provided it is conservatively decided that a partial removal may be begun, the skin-flap is raised, all of the subcutaneous vessel ligated, and a conclusion reached after this step whether or not it is safe to proceed. If it is considered unsafe, one inferior thyroid artery may be ligated and the skin-flap returned to its place and held by skin clips and the degree of reaction observed. If this is not excessive, the remaining inferior thyroid artery may be ligated in two to three days. If it is not unusual following elevation of the flap a right subtotal hemithyroidectomy may be done, and if doubt then exists, the wound is closed and the patient sent home for six weeks, following which the gain is so remarkable that the final left subtotal hemithyroidectomy may be done with almost absolute safety.

This, then, represents the entire list of multiple stage measures which, when viewed as a whole, appears indeed formidable. We have employed the entire seven-stage procedure but one time in our entire experience; the five- and six-stage in less than 1 per cent.; the four-stage in 6 per cent.; the three-stage in 20 per cent.; the two-stage in 35 per cent., and the one-stage in 38 per cent.

In support of these procedures we state that during the year 1921 we did 342 operations on 225 patients, with 3 deaths; during the year 1922, 450 thyroid operations on 321 patients, with 1 death; and during the year 1923, 584 thyroid operations on 430 patients, with 3 deaths—a total of 1376 operations on 976 patients, with 7 deaths.

In a group of 1258 operated cases within the last few years in our clinic, 583 were primary hyperthyroidism, with a mortality of 1.7 per cent.; 320 were secondary hyperthyroidism, with a mortality of 2.1 per cent., and the remainder were nontoxic goiter, with a mortality of 0.51 per cent.

We believe that the value of multiple stage operation as a trial procedure lies in the fact that it gives evidence of the degree of reaction and protects against the untoward reaction

which plays a major part in the mortality of the surgical treatment of toxic goiter, and that, furthermore, it is a measure which divides the burden upon these toxic patients who are often so ill prepared to endure it.

We believe that we occasionally do more operations on some individuals than perhaps might be necessary, but we know of no way of ascertaining whether or not a lesser number may be used except by jeopardizing the patient's life, and we prefer the moderate added discomfort to the possible added disaster.

Having accomplished what appears to be at present the minimum mortality, as is the case with the other large thyroid clinics, and having provided means for the differentiation of cases so closely simulating thyroidism, but of non-thyroid origin, we come now to the third and last phase through which we have progressed.

Early in our thyroid experience we were impressed, and in a considerable measure intimidated, by the cases of auricular fibrillation which complicated the already trying thyroid situation in many of our cases of exophthalmic goiter or hyperthyroidism.

We were led because of this to seek for our clinic the association of a physician particularly interested and trained in the subject of cardiology, and with the attachment of Dr. Hamilton to the clinic the surmounting of the difficulties of our third phase, so to speak, was accomplished.

We soon learned that with proper digitalization many of the toxic goiters could be restored to regular pulse-rates, and that even though absolute irregularity still persisted, a very great majority of these cases could be operated upon successfully and withstand the various surgical procedures on the gland with quite remarkable impunity.

It has been our experience that almost without exception the large group of patients who have recurrent attacks of fibrillation before and after operation return to normal rhythm and never fibrillate again after relief of their thyroid toxicity by subtotal thyroidectomy. On the other hand, a fraction of the group of patients with chronic constant auricular fibrillation

continue to have irregular heart action, and never return to normal rhythm. This small subgroup of this phase has been of interest only in so far as we have learned two things: one, that cases of auricular fibrillation withstand the surgical procedures on the thyroid quite well; two, that elimination of thyroid toxicity restores to permanent normal rhythm many cases, even those with irregular heart action which has persisted for months or years.

Our attention was directed then to the more important part of this phase by the observation of a gradually enlarging group of cardiac cases, in the beginning seen at their homes in varying degrees of cardiac decompensation, and with clinical features indicating the presence of thyroidism varying in apparent toxicity from confusing borderline mildness to obvious frankness. It was observed in these cases that in spite of appropriate measures, such as rest in bed, morphin, and cardiac stimulants, it was not possible to lower the pulse to within normal limits or to ever completely overcome the decompensation.

This led us to a careful study of this group of cases, now termed by us thyrocardiacs, both from the point of view of diagnosis, which is often difficult, and the operative relief, which is brilliantly gratifying.

The distinctive factors of the thyrocardiac group have been stated above, that is, that decompensation in this group of cases is in part brought about and in whole made persistently resistant to medical treatment by an underlying hyperthyroidism. The ready clinical diagnosis of this group without special study is made difficult by the fact that the obvious and urgent clinical picture, overshadowing the underlying thyroidism almost completely, is that of cardiac failure. It is further made difficult in the majority of the cases by the fact that the picture of thyroidism in this group of cases is atypical. The thyroid gland in many of the cases is not only not enlarged, but it is smaller than normal. The patients are often not activated, as is usually the case in toxic goiter. They are apathetic. They frequently show generalized bronzing of the skin. They appear much older than they are. They may retain the eye stigmata of past thy-

roidism, or may have no eye signs whatever. Their past history usually elicits the suspicion of a thyroidism. For example, nearly all of them have lost large amounts of weight unaccountably, although this may not be at once obvious because of the edema, and their basal metabolism rate remains persistently elevated.

From having carefully studied and operated upon a group of 52 such cases we have concluded that they represent an entity; that every case of cardiac failure with associated goiter should be critically studied to rule out the existence of thyroidism as the underlying cause; that any patient with decompensation and auricular fibrillation or with marked loss of weight and bronzing of the skin with or without thyroid enlargement should in the same way be studied with this point in mind. We believe, from our experience—in which we have seen men who were extremely able but inexperienced in this group of cases pass over and completely fail to recognize patients within the group—that there exist many of these cases now unrecognized and chronically disabled, but potentially curable.

We have learned that with proper preparation and care patients in this group, even though still in partial decompensation, may be operated upon. It has been surprising how well they have stood surgical procedures done either in one or two stages, hemithyroidectomy, or complete subtotal thyroidectomy. Up to the present more than two stages have never been employed. It is an essential part of treatment to eliminate toxicity immediately, since by no other means may the two factors which produce heart failure—rapid rate and absolute irregularity—be overcome.

We have now operated on 52 of these decompensated cases. One died suddenly following the operation, possibly from embolus; all others survived the operation; two with complicated heart disease were relieved for a time, but heart failure returned later, and they died. Two cannot be traced; all the others are relieved of heart failure. Of this number in the cases operated since January of this year it is too soon to predict how much they can do, but some have already returned to full duty and

others to partial duty. Thirty-three traced cases had been operated before January 1st of this year, and of these all are doing their particular full duty. They now average twenty months of full ability; before operation they averaged twenty-one months of disability.

Since discovering this group we have frequently stated that we know of no parallel situation in cardiology where by means of a surgical operation it is possible to remove in a relatively short time the burden tachycardia and auricular fibrillation which is so well calculated to produce decompensation in a heart the reserve margin of which is narrow.

We come now to what may or may not be the beginning of a further phase in the treatment of toxic goiter. I refer to Lugol's solution. We are as yet unable to make any hard-and-fast statements as to the effect of Lugol's solution. We have administered it to 62 cases of primary hyperthyroidism, it being our plan to give Lugol's solution to every fifth case of exophthalmic goiter in order that a critical comparison could be made.

From a review of the records and charts of these cases we feel justified in stating as follows:

Lugol's solution is not a method of curing hyperthyroidism. It is a method preparatory for operation.

A few cases receiving rest and Lugol's solution have shown striking drops in basal metabolism rate and clinical improvement, greater than we have ever seen with rest alone.

A number of cases have shown considerable drop in basal metabolism rate and clinical improvement, but the same drop in basal metabolism rate and clinical improvement has been noted in a few cases with rest alone.

A definite number of cases have occurred in which Lugol's solution has entirely failed, and in one case the drop in basal metabolism rate and apparent clinical improvement so misled us that we, for the moment, shed our cautious reservations as to possible untoward reaction, did too much, and the patient died an intensely toxic death in spite of the previous apparent improvement. From this and other cases showing marked but not fatal reactions we are of the opinion that the replacing of

preliminary surgical measures by the administration of Lugol's solution is fraught with serious possibilities unless it can be later proved that our experiences have been exceptions, and that the drop in basal metabolism rate and apparent clinical improvement are real and indicate an ability on the part of the individual to withstand procedures proportionate to his apparent condition.

Finally, we are firmly convinced that Lugol's solution should not be administered without a knowledge of its dosage by mouth and rectum and a knowledge as to how long it should be continued.

Its complete failure in such a very definite percentage of cases, having the same conditions of disease as those in which it is thought to succeed, should make us insist that it have a fair and impartial trial, but that no credit be attached to it except that which it can demonstrate, beyond measure of doubt, to have earned.

Having spoken so far of the surgical treatment of toxic goiter, it is our duty to express our sentiments regarding the two remaining methods of treatment—*x*-ray and medical treatment.

Regarding medical treatment, there seems to us little to say except that if it is persisted with there is always the possibility that one of the natural remissions of the disease may appear. We know of no drug which is able permanently to lower the basal metabolism rate. Rest in bed relieves the organs of a considerable burden, but a great many of the patients upon whom we operate have had long periods of rest in bed without avail, and many of these patients have also had temporary remissions of the disease and suffer from a renewal of the toxicity.

Each year, also, so far the medical mortality in the clinic—that is, the number of patients coming to the hospital vomiting and in imminent danger of death—has been greater than the surgical mortality.

Our views regarding the *x* ray treatment of toxic goiter are as follows:

We are opposed to the *x*-ray treatment of toxic goiter because it requires, at least, three months to ascertain whether or not it is to be effectual. A much lower percentage of cases are benefited by *x*-ray than by surgery, and in those cases benefited there is a much higher percentage of recurrence than with surgery. Our past experience with limited surgery on the thyroid gland, such as hemithyroidectomy, has resulted in recurrence of the disease in the remaining lobes even though what appeared to be a cure was obtained for a period of time by the original hemithyroidectomy. We have, therefore, been convinced that anything short of removal of four-fifths to five-sixths of the entire hyperplastic thyroid does not insure the patient a complete and permanent cure.

Since *x*-ray treatment possesses no indications of its success except those of obvious clinical improvement, we see no way by which the dosage can be so gaged that sufficient thyroid is removed to produce a permanent cure (four-fifths to five-sixths) and not produce myxedema. This we believe to be an explanation of the high percentage of recurrences. We further feel that in the three months required to ascertain the effect of *x*-ray treatment with its possible failure and possible recurrence, we can by surgery have the patient, in most instances, so relieved of his symptoms that he is almost able to return to work.

In conclusion, we feel that the ideal method of treatment for toxic goiter, with its possible serious complications and its ability to incapacitate patients, is that which most quickly, most completely, and most certainly restores them to the state in which they were prior to the onset of the disease, and the method most nearly meeting these requirements, in our experience, is surgery.

We feel that individualization and centralization of this group of cases is essential for success with them.

We believe that the group we have called thyrocardiacs have, in a large measure up to the present time, been overlooked, and that there exists a large number of these patients now undiagnosed who are potentially curable.

Hard-and-fast conclusions as to Lugol's solution should not be reached until one's experience with the treatment is considerable, but we see no disadvantage in administering it as a preparatory measure to operation in toxic cases of primary hyperthyroidism.

PRELIMINARY LIGATIONS IN THYROIDISM

FRANK H. LAHEY

IN a discussion of preliminary pole ligation one does well first to admit the uncertainties as to how it brings about improvement, uncertainties in a few cases that it will bring about improvement, and the fact that in a definite percentage of cases there is little or no material drop in the basal metabolic rate.

There is no tangible evidence now available as to why there is such improvement following pole ligation. It has been assumed that the limitation of blood-supply occasioned by ligation of the two superior thyroid vessels, for example, cuts down to some degree the ability of the gland to function. Such is the vascularity of the gland, however, that operations upon it following preliminary pole ligations find it in no way lacking a plentiful supply of blood.

It has been assumed also that the ligature which surrounds the superior thyroid vessels includes also the nerves from the superior cervical sympathetic ganglia, thus interrupting its conductivity and lessening the impulses coming to the gland along this tract.

This theory is practicable and possible, but in actual practice, unfortunately, it is not possible to be sure, first, that the nerve is included in the ligature, and, second, if it is, that it is the cause of the improvement.

It has been our experience in a large series of preliminary pole ligations that very often the metabolic rate is either not at all or not materially lowered following ligation, even at times when there is a definite gain in weight and improvement in general condition. Also in a certain small percentage of cases there has been little if any obvious clinical improvement. This we have been unable to explain to our satisfaction, yet in spite

of this and the above stated uncertainties attached to the procedure, we believe strongly in its application in extremely toxic goiter.

We have repeatedly stated and are still firmly convinced that a majority of doubtful operative risks are made better and safer when coming to partial thyroidectomy by a preliminary pole ligation. We feel strongly convinced that had we, for example, 100 patients all with such intense hyperthyroidism that we felt uncertain as to how they would endure an operation directly on the thyroid, those having preliminary pole ligation done first would show a lower mortality rate than those having any operation directly upon the thyroid gland. The former group we have, of course, in our clinic had opportunity to observe in considerable numbers, that is, the cases with preliminary pole ligation, but feeling as we do, it is obvious that we cannot have the latter group. It is unfortunate also that our ligation cases cannot be compared with the cases of some other group in which the first operation is one undertaken primarily on the thyroid gland. This is true because, unfortunately, there are no fixed groups into which cases may be relegated, such as single X toxicity, double X, triple X toxicity, and so forth. Thus all estimated degrees of toxicity and also those of operability must vary with the observer's viewpoint, experience, caution, and enthusiasm for a method.

That obvious clinical improvement follows preliminary pole ligation no one who has observed these cases in large numbers can fail to admit. This, however, is not the sole factor which has led us consistently to submit our cases of doubtful operability to preliminary pole ligations and partial operative procedures upon the thyroid gland. We hold to preliminary partial procedures in the surgery of severely toxic goiter also because of its test-out value as related to the sick thyroid patient's ability to withstand operative procedures, and this, we believe, to be of equal value with the general improvement gained by the procedure.

We have always maintained that possibly slightly excessive caution on our part might result in one, perhaps two, unneces-

sary preliminary operative procedures being done. One must admit, however, that this is distinctly less expensive to the patient and trying to the surgeon than the method whereby one is convinced, if only in a few cases, that one or two preliminary operative measures might have been superior to the complete procedure perhaps disastrously employed, or so dangerously close to it as to have been not worth the risk on the patient's part.

Lugol's solution which has come so into prominence of late may perhaps largely replace preliminary pole ligation (of this we are still much in doubt), but where uncertainty exists as to whether or not a patient will endure a subtotal thyroidectomy, a death following this operation should make the surgeon feel that the more cautious approach through preliminary procedures would have lessened the danger of such a fatality. What, after all, is one's primary duty? To save the individual from repeated operative procedures, or to make most certain that he endures the complete surgical ordeal the most safely?

We wish to maintain still that when any operative doubt exists, even though the patient has been thoroughly prepared with Lugol's solution, preliminary therapeutic and test-out surgical procedures should be employed.

We strongly urge, with no reflection whatever on the method of preparing patients for operation with Lugol's solution, that any elimination of preliminary surgical procedures in the operative treatment of severely toxic goiter be arrived at solely on the basis of cautious individualistic experience, and not upon enthusiasm for a new, novel, and hopeful procedure, however auspicious it may appear.

Regarding the technic of superior pole ligation, we have maintained that if the patient is submitted to a preliminary surgical procedure, such as pole ligation, because the complete operation is too risky, then the lesser procedure is unjustified unless the lesser operation is so accomplished technically that all of its possible benefits may be assured the jeopardized patient. For which reasons, therefore, we have insisted in this clinic that the operative procedure shall be so planned that the

entire pole is exposed, so that one is certain that all of the pole is entirely surrounded by the ligature, making absolutely sure that all of the vessels are *within* the grasp of the ligature, and making as sure as possible that the nerve also is included.

The technical steps of superior pole ligation are, then, the demonstration of the common carotid artery and internal jugular vein opposite the level of the upper pole of the gland, the

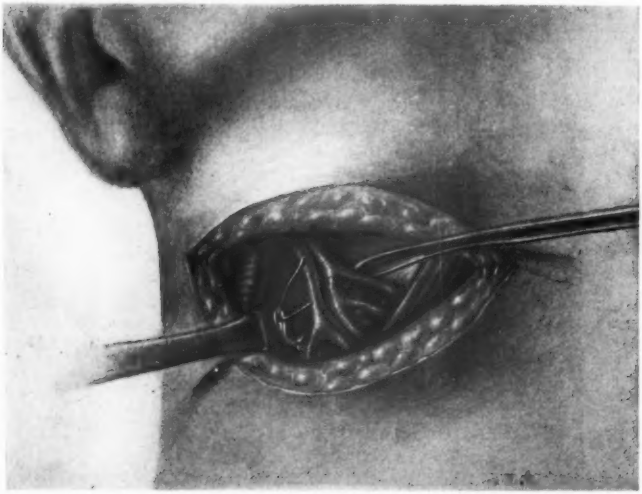

Fig. 519.—Note the retraction of the great vessels and the method of passing the special carrier downward along the thyroid cartilage. The incision has been made unduly large in order to demonstrate the anatomic relations.

retraction of these vessels outward, the retraction of the oblique fibers of the omohyoid muscle inward, and the complete exposure of all structures entering the upper pole of the gland. By means of the special carriers shown in Fig. 519 the passer may be carried down along the external plate of the thyroid cartilage well back behind the posterior aspect of the entering vessels, and safely out beside the retracted great vessels. By this method one may be sure that the ligature entirely surrounds the

pole. Since the great vessels commonly lie in close contact with the upper pole, it has been our experience that only by this maneuver of retracting them may one be certain that the ligature under direct vision entirely surrounds all entering structures without injury to the common carotid artery, vagus nerve, and internal jugular vein.

THE TECHNIC OF ANESTHESIA FOR THYROID OPERATIONS

LINCOLN F. SISE

IT is desirable that the anesthetic for thyroid cases, especially the toxic cases, disturb the patient as little as possible. It is not a question here, as in many operations, merely of discomfort to the patient, but because a postoperative reaction may be started by any sort of strain or disturbance it may, with these patients, be a question of life or death.

Of the anesthetics which have been generally used, ether disturbs the patient more than any other. It has a definite effect on the tissues of the body, it upsets the acid base equilibrium, it has a tendency to produce nausea and vomiting, and it is eliminated slowly from the body. Clinical experience shows that postoperative reactions are more prone to occur after this anesthetic than they are after some others. While of entirely secondary importance, yet the fact that induction is longer than with other anesthetics may be worth consideration in a busy clinic.

Ethylene appears to be much more desirable than ether, as it has practically no systemic effect. In some ways it is more desirable than nitrous oxid, as no cyanosis accompanies its use, and, consequently, there is probably less rise in blood-pressure. Its extreme inflammability is a very serious drawback, and is the reason it is not being used in this clinic.

Local anesthesia does not of itself disturb the patient. When properly given it has practically no effect on the tissues of the body beyond the points of injection. But, unfortunately, the patient remains conscious, and this fact frequently subjects her to a considerable nervous strain. It is this fact mainly which has caused us gradually to abandon its use in favor of nitrous oxid-oxygen.

Local anesthesia combined with nitrous oxid-oxygen anesthesia has not, in our hands, showed any noticeable lessening in the amount of nitrous oxid necessary, nor any other noticeable benefit such as to make its use seem advisable.

Nitrous oxid-oxygen has proved itself with us to be the most satisfactory routine anesthetic for these cases. Except for the concomitant oxygen lack its systemic effect is practically nil, and with the technic which we use the oxygen lack is relatively slight. Induction and recovery are quick and not unpleasant. Thus patients do not dread the anesthesia when multistage operations are necessary. Our clinical experience is that the postoperative condition and appearance of patients who have had nitrous oxid-oxygen compare favorably with that after other anesthetics.

In certain types of patients even a slight oxygen lack is undesirable. Such patients are the very weak and exhausted, those who have lost weight rapidly or in high degree, and particularly those with an unsound heart, perhaps from rheumatic disease or sclerotic changes, and especially those having auricular fibrillation. In all these cases even a comparatively slight oxygen lack may give rise to danger of sudden death under anesthesia. This danger from lack of oxygen may be eliminated by the addition of an amount of ether which is at once comparatively small and at the same time enough to make proper oxygenation of the patient possible.[1]

Our scheme of anesthesia is, then, the use of nitrous oxid-oxygen as ordinary routine, and the addition to this of ether in minimal amounts whenever it is indicated.

Special methods of anesthesia are rarely necessary. On one occasion, in a case of lingual thyroid, we have administrated ether by the intratracheal route in order to avoid inhalation of blood.

Any of the better types of nitrous oxid-oxygen apparatus are probably satisfactory. In this clinic we have used mainly

[1] We have just started the use of ethylene in these cases. While the number of administrations is too few to draw conclusions, yet these few cases seem to indicate that ethylene is a great improvement.

the type with the breathing bag next to the patient. This bag is small and oblong and can be turned to one side by means of a slip joint in a curved chimney piece, so that it does not get in the way of the anesthetist. The mask fastens more easily to the face when the leverage of a hose does not also have to be held. The bag near the patient is quite useful in obstruction, as will be described later. It may also be useful if the trachea is accidentally cut. If this happens, and if there is no obstruction between the breathing bag and the cut, positive pressure may be kept up in the trachea during inspiration by means of pressure on the breathing bag, and the inhalation of blood thus prevented.

The usual face masks have a chimney, or valve, coming up from the top and, consequently, are so high that they get in the way of the operator. The one used here has a smooth, low top, with an exit to the bag just above the nose. At the exit there is a slip-joint where a slightly curved chimney piece, about 4 inches long, leads to the breathing bag. A clean mask can be slipped on this chimney piece for each new patient. By means of the slip-joint the chimney piece can be turned to one side or the other as needed to get the bag out of the way.

A Boothby collar, somewhat modified, is used to hold the mask in place. This was originally designed by Boothby to be used instead of an inflatable rubber face-piece; but we have found it here very useful *in addition* to such a face-piece. It has some advantages over the more usual method of holding the mask by means of a towel clamped over it. This collar can be adjusted after the sheet has been thrown back over the head and the operative field exposed. We have found it almost impossible to do this with towels, without throwing the sheet down in the operative field and disturbing the operator. It allows a better view of the patient's face. While it may not be quite as firm as the towels, it is sufficiently so in this type of machine where a hose does not run directly to the mask.

The blood-pressure is recorded throughout all operations. In an ordinary blood-pressure cuff one outlet is stopped and the other is led by a tube to a Y at the head of the table, one arm

of which goes to the dial and the other to the bulb. A ring in the edge of the cuff holds the bell of a Bowles type stethoscope in place, and a tube leads from this to ear pieces at the head of the table.

A chart holder and charts also are fastened at the head of the table. A chart is ready for each patient, and on it are recorded all important preoperative findings, to be mentioned later. Charts of any previous operations are also on hand.

Fig. 520.—Operating-table in the position used during thyroid operations. The upper half of the body is raised at an angle of about 20 degrees, and the thighs are flexed, so that the patient shall not slip down on the table. The bar used for extending the neck is shown covered by a pad. The blood-pressure cuff lies on the stool.

After the patient is placed on the table she is moved, by breaking the table, so that the head and trunk are elevated to an angle of about 20 degrees and the knees are somewhat bent (Fig. 520). A bar is placed under the shoulders which can be raised to any desired height. The patient's proper position on this bar is quite important. It should be placed just along the upper line of the shoulders (Fig. 521). When the bar is raised this arches the neck forward in the region of the thyroid and in the great majority of instances gives an excellent position (Fig. 522). If the bar is placed too high on the patient the head will not

Fig. 521.—Patient in position on bar before bar is raised. Blood-pressure cuff is on arm with tubes leading to dial and ear-pieces at head of table.

Fig. 522.—Same as Fig. 521, but with bar raised, showing extension of neck.

extend, and the upper part of the neck is raised instead of being lowered, and the desired arching is not obtained (Fig. 523). If it is placed too low the region of operation rises and falls

Fig. 523.—Bar placed too high on the patient, showing that no extension of neck is obtained. The bar is under the neck, but the perspective makes it appear to be placed higher than it really is.

Fig. 524.—Draping the patient (1): A half-sheet is passed under the head, and the ends are fanned out with one corner on each side coming down over the trunk.

excessively with respiration, and frequently the proper arching is not obtained. If it is *raised* too high the back curves, and

the bar is then apt to slip up and become placed too high on the patient. If it does not slip up the extreme extension of the head and arching of the neck which result, while giving excellent exposure, are very apt to cause respiratory obstruction.

Patients are draped for the operation in the following manner: A half-sheet is passed under the head, and the ends are fanned out with one corner on each side coming down over the trunk (Fig. 524). Two towels are laid on the upper part of the

Fig. 525.—Draping the patient (2): Two towels are laid on the upper part of the chest so as to make a V, with the point down and the two arms coming up over the shoulders on each side of the neck, and are clipped together at the point of the V.

chest so as to make a V, with the point down, and the two arms coming up over the shoulders on each side of the neck, and are clipped together at the point of the V (Fig. 525). Another half-sheet is now laid over this V, with its upper edge at the throat. This edge is clipped to each arm of the V together with the first half-sheet on each side of the neck (Fig. 526). The free part of the second half-sheet is then thrown up over a wire hoop which has been placed over the patient's head. The operative field is now excellently exposed, but entirely sepa-

rated from the field of the anesthetist (Figs. 527, 528). The draping is firm and not easily disturbed, and yet no clips have been caught in the patient. Thus it can also be used when local anesthesia is employed.

Before operation patients receive a preliminary narcotic. With the great majority of patients this consists of morphin $\frac{1}{4}$ grain and scopolamin 1/200 grain two hours before operation, and again one hour before, making a total dose of $\frac{1}{2}$ grain of morphin and 1/100 grain of scopolamin. This puts the patient

Fig. 526.—Draping the patient (3): A second half-sheet is laid over this V with its upper edge at the throat. This edge is clipped to each arm of the V together with the first half-sheet on each side of the neck.

in a drowsy condition, so that preoperative apprehension is greatly lessened. Anesthesia is easier and better, less anesthetic is required, and there is less cyanosis. Even in the few cases in which excitement results from the narcotic the anesthesia remains the same. After operation there are usually several hours of quiet sleep. Occasionally a patient gets complete loss of memory for the entire procedure. She goes to sleep in her bed, and wakes in it, without at first realizing that there has been any operation.

In a few instances excitation may result from the narcotic

Fig. 527.—Draping the patient (4): The free part of the second sheet is then thrown up over a wire hoop which has been placed over the patient's head.

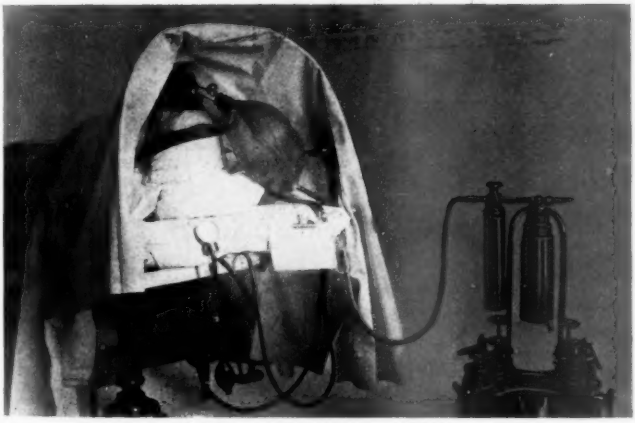

Fig. 528.—The anesthetist's end of the table, showing the easy access to the patient and the complete separation from the field of operation. Note the low mask held on by the Boothby collar and the oblong breathing bag turned to the right well out of the way. Just to the right of the bag is a rest for the operating nurse's elbow. Below the bag are the anesthetic charts in their holder, and to the left of this is the dial of the blood-pressure apparatus.

instead of quiet. The patient is restless and talkative. This may even go on to actual delirium. These two conditions are not usually seen in the quiet of the bedroom, but are started when the patient is disturbed by being transferred to the truck for the start to the operating room. They end with the administration of the anesthetic, do not seem to have any noticeable effect on the anesthesia, and do not usually reappear after the operation and return to bed.

In toxic cases, and in a large number of non-toxic, the pulse-rate is raised on giving the first hypodermic. In a few cases the pulse-rate is lowered by the narcotic. Lowered respiratory rate is also occasionally seen, but never of sufficient degree to be of importance, unless there is marked obstruction to respiration. In such cases the narcotic should be reduced or administered with great caution, and the patient should be closely watched.

The second dose of scopolamin is omitted if there has been an undue rise of pulse after the first dose, or the whole second dose, morphin as well as scopolamin, may be omitted in individuals of the weaker type who do not appear to be apprehensive. In individuals of the apathetic type the scopolamin is omitted entirely and the morphin may also be reduced in proportion to the condition of the patient. The scopolamin is also not infrequently reduced or omitted in thyrocardiacs, particularly the older ones.

All patients are seen personally by the anesthetist before operation in order that he may have a first-hand impression of their condition. He then goes over the recorded clinical history, the findings of the cardiologist, temperature chart, and laboratory examinations. He also assures himself that there are no respiratory infections. This is particularly important where the goiter is intrathoracic, as here there seems to be a slightly increased risk of pneumonia. In determining the question of infection a temperature between 99° and 100° F. does not appear to be of great significance in toxic cases.

In making the induction it is well to go somewhat more slowly than in patients who have not had such a preliminary narcotic. A too rapid induction may induce undue depression

of respiration and circulation. This is the same phenomenon as may be seen with any patient during induction, but it comes on more readily with these patients, who either have had a considerable preliminary narcotic or who are in a somewhat weakened condition. No trouble is experienced if the induction is a little slow, and in any event no trouble is experienced throughout the rest of the anesthesia.

False teeth may usually be left in place except when they are small, partial sets. The lower jaw does not become relaxed, but remains firmly set against the upper, so that there is no danger of the teeth slipping back into the throat; and the presence of the teeth makes the fit of the face-piece better and prevents obstruction due to blowing in and out of the lips and cheeks.

Laryngeal spasm occurs during thyroid operations just as it may in the course of any other anesthesia. It is probably due to the light anesthesia, together with the lodgment of a small amount of mucus or saliva in the glottis, and possibly to irritation from operative manipulations in the region of the larynx. It is seldom enough to cause more than slight inconvenience. On but one occasion in this clinic has it been of sufficient intensity and duration to cause alarm. It should be treated by the exhibition of oxygen in proportion to the severity of the spasm, and after a variable length of time it will lessen and disappear.

Obstruction to respiration occurs in goiter cases somewhat more frequently than in most other operations. Chief among the causes are position of the neck, pressure of a tumor, operative manipulations, falling of the tongue into the throat, and relaxed alæ nasi. In some individuals the arching of the neck produces obstruction, which is quickly relieved by bringing the head forward. In such cases a compromise must be made between the relief of the obstruction and the need of the surgeon for exposure. In those cases which have before operation been most severely obstructed by tumor local anesthesia has generally been used. Consequently, under general anesthesia, severe obstruction is not seen from tumor pressure, unless it is added to by the manipulation of the surgeon. In this case, if

the obstruction becomes too severe or prolonged, he can reverse his procedure and relieve the obstruction. In these two forms of obstruction and in that caused entirely by manipulation of the surgeon a considerable amount of relief may be had by using the breathing bag for artificial respiration. When the trachea is obstructed the patient's inspiration produces negative pressure below the point of obstruction, and this tends to collapse the trachea still further. Her expiration produces positive pressure, and tends to dilate the trachea and force air past the point of obstruction. Expiration itself is also considerably more powerful than inspiration. Thus the chief difficulty in these cases of obstruction comes with inspiration. If moderate pressure is made with the hand on the breathing bag during inspiration positive pressure is produced in the trachea above the point of obstruction, the trachea is dilated as during expiration, and the gases are forced past the point of obstruction.

The last two forms of obstruction mentioned above, those from the tongue and from the alæ nasi, are rather unusual. They may be treated as in any other anesthesia, if at all severe, by means of nasal tubes. In the last case the tube needs to be only long enough to enter the vestibule of the nose.

In cases of severe exophthalmos there is distinct risk of ulceration of the cornea; and this ulceration, once started, may go on to the production of complete blindness, in spite of all that can be done. As these prominent eyes frequently remain partially or wholly open during anesthesia, it becomes of great importance to protect them from an abrasion of the cornea, which might be the starting-point of a destructive ulceration. The eyes should never be covered by any material such as a towel or gauze which, if the eyes opened, might cause an abrasion. If they remain open at all they should be filled with sterile vaselin to prevent drying and to lessen the abrasive power of any chance contact; and they should be watched constantly to see that this chance contact does not occur.

Watching the condition of the patient and determining from it the extent of operation which is justifiable is the most difficult and, at the same time, the most interesting duty which falls

to the anesthetist. He should have before him, preferably on his anesthesia chart, all findings of the case which may help him in this determination, such as the salient features of the patient's history, her age, weight loss and disability, her physical and laboratory findings, heart and kidney condition, weight, metabolism, and any other condition which may have a bearing on the situation. He should also have the records of any previous operations, if multiple operations have been necessary, showing the extent of the operations, the course of the anesthesia, and the postoperative reactions. He should note the general appearance of the patient and depth of anesthesia and should record at frequent intervals the pulse-rate, blood-pressure, and respiration rate. All these have a bearing on the decision to be arrived at, and only by a correct evaluation of all these various elements can a correct decision be reached.

With non-toxic patients the same general principles apply in determining the extent of operation as in any other surgical operation, and, consequently, these need not be discussed here. With toxic patients there enters a new factor, which is not present with other patients and which complicates the determination of their condition. This new factor is the possibility of the occurrence of postoperative thyroid reaction. To aid in making the determination toxic patients may, entirely apart from their toxicity, be divided into two main groups—a sthenic, activated group comprising the great majority of patients, and an asthenic, apathetic, and exhausted group, comprising the small remainder of patients. These two groups present distinctly different indications during anesthesia. Thus they should be definitely distinguished one from the other before the beginning of an operation.

With the activated type of patient anything indicating an increase in activation, such as increased pulse-rate, pulse pressure, systolic blood-pressure, respiration rate, or a decrease in diastolic blood-pressure, tends to indicate a shortened operation. These patients seldom exhibit exhaustion during operation, as evidenced by the combination of rising pulse-rate and falling blood-pressure, and then only during the longest opera-

tions or where an unusual amount of blood is lost. They sometimes show a bounding, snappy pulse, which is indicative of potential reaction. In judging the height of readings, time should be allowed for the subsidence of any excitement which may have arisen during induction.

The apathetic type of patient makes up a small group which, in this clinic at least, has furnished the bulk of the immediate surgical mortality not due to surgical accidents, such as infection, embolism, etc. They should, therefore, be treated with the utmost caution and should usually be done in multiple stages. They usually come to the table more deeply flushed and more deeply asleep than the other type of patient would with the same preliminary dose of narcotic. Their anesthesia is colorless and gives no suggestion of a serious outcome. The readings are moderate or low. Even the metabolism fails to give a warning, as this also is usually low. Only the small dose of narcotic and of anesthetic necessary to produce respectively sleep and a sufficient depth of anesthesia give any warning that all is not well.

It is evident, then, that if we find our anesthesia is progressing in this colorless and apparently favorable manner we must be definitely sure that this apparently favorable course is due to the patient's good condition and not to her exhausted condition, that she is of the activated type and not of the apathetic type, for those of the activated type who come to operation with slight toxicity often show the same moderate or low readings as the exhausted cases of the apathetic type. If, therefore, a patient cannot before operation be definitely determined to be in good condition, and if during operation she runs this colorless anesthesia with rather low readings, she should be regarded with great caution.

The time element, which is of some value in all thyroid operations, is of special value in the toxic cases, but is of the greatest importance in this group of apathetic patients. The operations should all be short. In a few instances the character of the respiration, a jerky or sobbing type, shows that the patient is not standing the operation well.

In making these decisions as to the extent of the contemplated operation a conservative stand is always taken. If there seems any real possibility that a severe reaction may result from a given operation, a lesser procedure is adopted, for if less is done than might have been done, more can always be added; but if too much is done, it is impossible to subtract any.

At the conclusion of an operation the patient should be awakened and requested to speak before she goes to her room, in order that the integrity of the recurrent laryngeal nerves may thus be demonstrated. After convalescence and before she leaves the hospital the amount of her postoperative reaction should be entered on her anesthesia chart so that it may be available if subsequent operations are necessary.

The main contraindications to nitrous oxid-oxygen which we have established here are the higher grades of respiratory obstruction, and the sickest of the thyrocardiacs and apathetics.

When nitrous oxid-oxygen is contraindicated, local anesthesia is chosen. Under operative conditions the head of these patients is in a rather small and somewhat enclosed space, and it is found that all, but more especially the thyrocardiacs and those with obstructed respiration, much appreciate a free supply of air. This can be given them by fanning, but much more easily and efficiently by means of a hose running from air pressure. By means of a free stream of air passing over the face many a patient is kept quiet and reasonably comfortable who would otherwise have a most uncomfortable and restless time, and who might, consequently, interfere seriously with the operation.

THE TREATMENT OF ADENOMATA OF THE THYROID

FRANK H. LAHEY

ADENOMATA of the thyroid must be considered as encapsulated masses of thyroid tissue possessing the ability to function just as does the thyroid tissue of the gland itself, capable of hypersecreting and producing toxic symptoms just as can the oversecreting entire thyroid gland in exophthalmic goiter.

Adenomata of the thyroid are submitted to surgical treatment in this clinic for any one of the following reasons: When they are unsightly, when they are producing pressure, when there is danger of malignant degeneration, when they are substernal or intrathoracic, when they tend to become substernal or intrathoracic, and when they are producing toxic symptoms.

Under the heading of "unsightliness" we have considered any adenomatous goiter disfiguring when it is of sufficient size to be unduly conspicuous. Unilateral goiters have been the most disfiguring type with which we have had to deal, and we have found single adenomata particularly prone to produce unsightly disfigurements in the neck. A very large proportion of the disfiguring goiters in our experience have been non-toxic and, therefore, their unsightliness has been endured, in most instances unnecessarily, since they could often have been removed very much earlier than they were with almost absolute safety.

We have seen obstructive pressure from goiter arise particularly in connection with adenomata and less often with cysts. Pressure has been most often upon the trachea, occasionally on the posterior wall of the laryngeal cartilage, and rarely upon the esophagus. We have occasionally seen colloid adenomatous goiter occur in prolonged upper poles of the thyroid gland, the poles turning inward beneath the superior thyroid vessels and passing behind the larynx on one or on

both sides (Fig. 529). In the latter case an encircling type of goiter occurs. We have rarely seen marked pressure result from superior thyroid poles occupying an encircling position behind the larynx, but, as a rule, have discovered them casually, while dislocating the pole in the course of a thyroid operation done for some other cause.

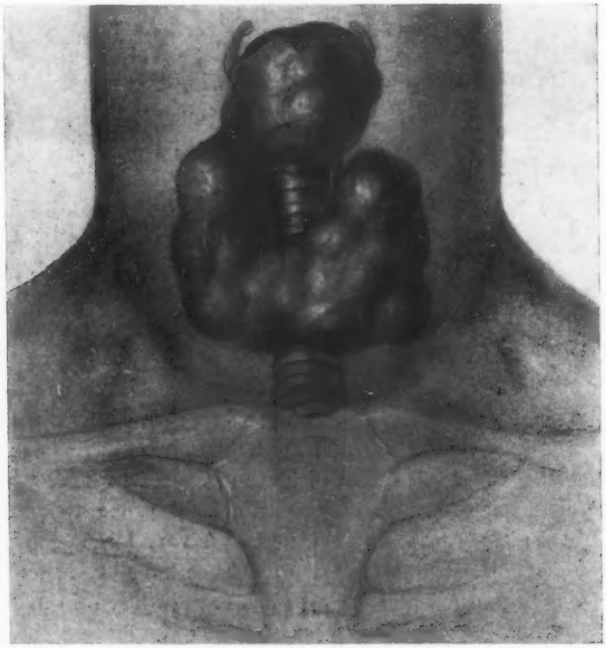

Fig. 529.—Showing adenomatous goiter which has slipped beneath the larynx.

Adenomata above the level of the sternal notch most often occur in one or other of the lobes, and as they enlarge they deviate the trachea either to the right or to the left as shown in Fig. 530. As the enlargement progresses and pressure increases the resilient tracheal rings are gradually pressed inward, until there may be complete flattening of the trachea on one side, so that the breathing space is represented solely by the curve in

the rings on the opposite side. Occasionally adenomata occur on both sides, so that lateral pressure upon the trachea is made from either side, until the trachea is converted into a mere slit of the so-called scabbard type. We have recently operated such

Fig. 530.—Two types of tracheal pressure are depicted here—antero-posterior flattening from a retrotracheal goiter, and lateral flattening and deviation of the trachea from an intrathoracic goiter.

a case in which the trachea was so collapsed that it was difficult to find a space through which to do a tracheotomy.

When adenomata, even of small dimensions, arising in the body or the isthmus of the thyroid have become attached to the trachea we have often been surprised to note the degree of tracheal denting which can occur with small tumors.

The most marked degrees of tracheal obstruction, other than those where bilateral adenomata collapse the trachea from bilateral pressure, have been seen in intrathoracic goiter, where

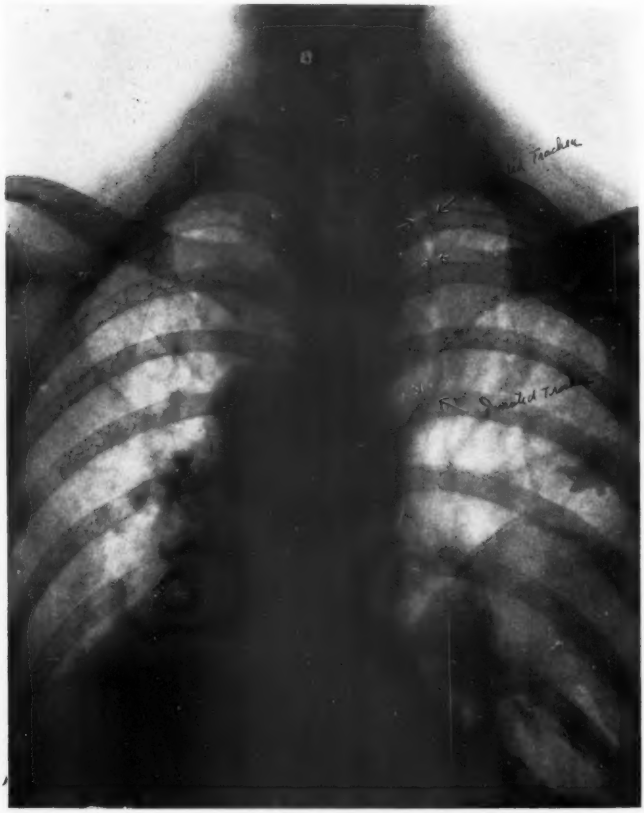

Fig. 531.—Note marked deviation and narrowing of the trachea due to intrathoracic goiter. Trachea shown deviated to the left as a light shadow.

a laterally located adenoma dislocates the trachea in a marked degree to one side or the other, causing high grades of tracheal obstruction, as shown by *x*-ray in Figs. 530, 531. Since pressure softening of the tracheal ring occurs so readily, and since respira-

tory obstructions play such a marked part in postoperative mortality, we feel that it is essential to remove adenomata in their earliest stage when no degree of tracheal pressure is present.

We have seen a few cases where intrathoracic prolongations of adenomatous masses have extended behind the esophagus, but we have never seen a case with esophageal obstruction.

Regarding the possibility of malignant degeneration as an indication for operation in thyroid adenomata we feel that if it is assumed that all breast tumors are surgical, it should, at least, be considered that thyroid tumors, too, are surgical, since evidence of malignant degeneration in tumors of the female breast is much more obvious than it is in the thyroid. If discreetness of outline be lost in adenofibroma of the female breast, it may be easily recognized by palpation, since the adenoma is placed in the soft surrounding tissue which lends itself readily to palpation. Adhesions to the pectoral fascia or to the overlying skin act as a warning indication, and if malignancy be discovered the organ lends itself well to radical removal together with the adjacent lymph-gland.

Adenomata of the thyroid, on the other hand, are rarely diagnosed as having become malignant until the process has been present for some little time. They do not lend themselves well to palpation. If malignancy has penetrated the capsule of the thyroid it, as a rule, has penetrated the thyroid gland, has involved neighboring structures, has metastasized into the deep cervical lymph-gland, and in a majority of instances is beyond hope from surgical removal. Such a case is best treated palliatively by *x*-ray.

In a review by Dr. S. M. Jordan, of this clinic, of 1675 patients sent to the metabolism laboratory, 1.2 per cent. were found to have malignant goiters. Now, if one realizes that 1.2 per cent. represents the percentage of malignancy in a group of cases some of which did not have thyroidism, but were sent to the metabolism laboratory for help in diagnosis, and that some of them had only exophthalmic goiter, it must be realized that the percentage of malignancy occurring in cases with true adenomatous goiter,

if it were estimated, would be found to be much higher than the figures stated above.

Because of this fact we have assumed that the best treatment of adenomata, which may not only become malignant, but are prone later to show toxicity,[1] is removal, when the patient approaches the age when malignancy is most apt to occur. We, therefore, advocate the removal of adenomata in patients of forty or over.

Adenomata and cysts of the thyroid represent almost all of the types of goiter which become intrathoracic in location. Intrathoracic goiters, as a rule, originate as adenomata located in the lower pole of the thyroid gland. These adenomata, it must be recalled, are covered by the prethyroid muscles, the sternocleidomastoid, sternohyoid, sternothyroid, and omohyoid muscles, largely attached to the sterno, longitudinal in direction, causing pressure anteroposteriorally on the tumor, and tending to direct its movement in a downward direction into the mediastinum. This tendency is further augmented by the ascent and descent of the thyroid on swallowing, and by the fact that there is no obstruction to prevent the adenoma passing downward through the superior thoracic aperture into the mediastinum. These are the factors which produce intrathoracic goiter, and when once the adenoma becomes located in the mediastinum it soon, if it continues to grow, becomes too large to escape readily through the superior thoracic aperture (Fig. 531). It is located on either side of the trachea and deviates that structure, as stated above, markedly to one side, often collapsing it so that stridor and respiratory difficulty results.

Cysts of the thyroid are quite commonly considered under an entirely separate heading in thyroid classification, and so naturally assumed not to possess the danger of malignant degeneration which adenomata do. Nearly all cysts of the thyroid were originally thyroid adenomata whose central tissue has become devascularized, necrotic, and finally liquefied, the original fibrous capsule of the adenoma representing the fibrous cyst wall.

[1] Of the adenomata coming to our clinic 42.1 per cent. are toxic.

Examination of cyst walls, however, will often show that this process of liquefaction is frequently incomplete. Islands and, often, complete layers of live, vascularized, glandular tissue remain attached to the inner wall of the fibrous capsule. These elements have potentially the possibility of malignant degeneration, therefore it cannot be assumed that cysts of the thyroid are free from the danger of carcinomatous changes.

We believe that intrathoracic goiters should be removed as early as possible, since they tend to produce tracheal pressure, and owing to sudden changes in their size, secondary to changes in their blood-supply, they may produce acute emergencies due to increased tracheal pressure, which may even go on to such a degree of suffocation that death results.

The removal of adenomata from the superior mediastinum necessitates drainage of the large cavity which is left in the mediastinum, and always exposes the patient to the possibility of mediastinitis, 2 cases of which have occurred in our experience, both resulting fatally.

Further, when distortions of the recurrent laryngeal nerve have occurred with large intrathoracic adenomata, one can never be certain that complete recurrent laryngeal paralysis may not occur as a result of their dissection and delivery.

For the above-mentioned reasons, therefore, we believe that all adenomata which are low lying and tend to become intrathoracic in location should be removed early before they do so, when the operative risk is considerably less and the possibility of complications markedly diminished.

Intrathoracic goiters are to be suspected in any patient in whom both inferior poles of the thyroid cannot be accurately palpated. They are to be suspected when there is dulness over the upper part of the chest, when there is obvious deviation of the trachea, and when there is unexplained respiratory difficulty, particularly in the presence of goiter. They rarely occur without producing deviation of the trachea. The *x*-ray will demonstrate their location (Fig. 531) and also the caliber of the trachea, showing not only the degree of dislocation but also the effect of pressure as well.

The mortality of removal of intrathoracic goiter is by no means high; the outcome is extremely satisfactory, and in an experience now of considerably over 100 cases, 6 of which were completely intrathoracic, it has been necessary to split the sternum in but one case.

Those adenomata which have existed benignly for some time, showing superimposed toxicity later on, we have termed "adenomata with secondary hyperthyroidism."

In the beginning of this discussion it was stated that one must consider adenomata as being encapsulated areas of thyroid tissue, capable of producing secretion just as the true thyroid tissue does. In secondary hyperthyroidism it must be realized that the hypersecreting tissue is that within the adenoma or adenomata, and that in many instances the thyroid tissue itself is secreting normally. This has been proved clinically by the fact that simple removal of adenomata or adenoma, no thyroid tissue itself being removed, has resulted in complete relief of symptoms, lowering of pulse-rate, gain in weight, and a return to normal in every way, a previously elevated metabolism rate being also reduced to normal. Clinically this is convincing evidence in favor of the above-stated assumption, since it is known that in primary hyperthyroidism or exophthalmic goiter it is not possible to cure the disease surgically except by removal of four-fifths to five-sixths of the entire thyroid gland. While, as stated above, in secondary hyperthyroidism or thyroidism secondary to toxicity arising from the thyroid tissue within the adenoma, it is possible to cure the patient solely by the removal of these adenomata.

To our minds primary hyperthyroidism and secondary hyperthyroidism, although occurring at different ages, as a rule produce the same signs and symptoms and differ only in the method of surgical treatment.

It should be realized always, however, in connection with these cases that the presence of adenoma or adenomata does not always indicate that the toxicity is arising from the hyperactivity of the adenomatous thyroid, since the adenomata may be existing only coincidently in a thyroid which is itself hyperactive.

The relief of secondary hyperthyroidism by excision of the toxic adenomata results in one of the most satisfactory cures occurring in all of the thyroid diseases.

Conclusions.—Thyroid adenomata should be treated surgically when unsightly, when producing pressure, when intrathoracic or tending to become so, in women over forty, to insure against malignant degeneration, and when producing secondary hyperthyroidism.

ABERRANT GOITER

FRANK H. LAHEY

THERE is little question now that the thyroid originates from a single anlage. Thyroid tissue has not been demonstrated arising from the ultimobranchial bodies, the presumed

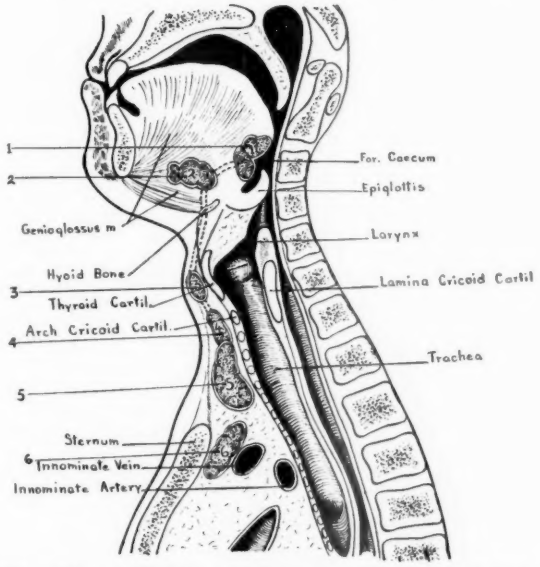

Fig. 532.—Diagram showing places where accessory thyroids may develop. Course of thyroglossal duct shown by dotted lines: 1, In the region of the foramen cecum; 2, intralingual; 3, prelaryngeal; 4, pyramidal process; 5, normal thyroid; 6, accessory retrosternal thyroid.

lateral anlagen of the past; moreover, the development of myxedema following the removal of a lingual goiter, which has happened in such a case in our own hands, clinically refutes such

a possibility. If a lingual goiter represents only the thyroid tissue arising from a median anlage, the tuberculum linguale mediale, then, even though this be removed, myxedema should not result if lateral anlagen exist, because they should have developed thyroid tissue which would prevent it. See case reported by us in Surg., Gynec., and Obstet., March, 1923, pp. 395–397.

Fig. 533.—Allied accessory thyroid. Note the extruded adenoma still attached to the thyroid by fibrous bands. (Diagrammatic.)

If it be accepted, then, that there exists a median anlage only, its point of origin is represented in the adult by the foramen cecum. A majority of aberrant thyroids exist from this point to the normal location on the tracheal rings; these are due either to failure of the entire thyroid to descend, or to segments of thyroid being left behind in the course of its descent.

Most of the aberrant thyroids, therefore, are situated be-

tween the hyoid bone and the foramen cecum at the base of the tongue, since few thyroids fail to make their complete descent if they pass below the level of the hyoid. Those which remain at the foramen cecum are the true lingual goiters (Fig. 532); those within the tongue, the intralingual goiters; those below the tongue, the sublingual goiters; those in front of the larynx, the prelaryngeal goiters.

Fig. 534.—Pseudo-accessory thyroid. Note the isthmus of true thyroid tissue joining the mass to the thyroid. (Diagrammatic.)

Two other not uncommon locations for aberrant masses of thyroid tissue are: one, within the superior mediastinum, the other just outside the sternomastoid muscle in the posterior triangle of the neck. Since neither of these locations is in the course of thyroid descent, it must be presumed that these aberrant masses represent separated elements of the thyroid organ, which have remained at one or other of these locations as rests, to be stimulated to later growth.

A few cases of intratracheal masses of thyroid tissue have been reported.

These masses of aberrant thyroid tissue, with the exception of the lingual forms, are of clinical interest solely because it is important to distinguish them from other tumors which may require removal.

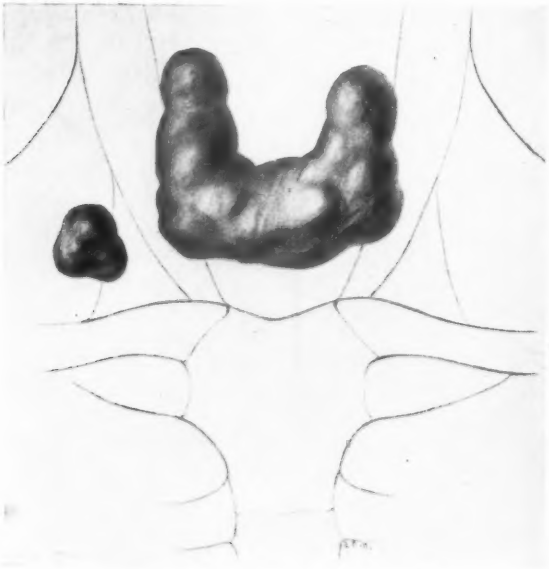

Fig. 535.—True accessory thyroid. Note the entire absence of any connection with the thyroid. (Diagrammatic.)

Lingual goiters, on the other hand, as in the case reported by us, are often of such size as to produce marked interference with swallowing, thus necessitating immediate surgical attention.

Surgically, the true lingual goiters are best approached through the mouth; the intralingual and sublingual ones through the floor of the mouth. When exposed at the time of operation, however, the true and false aberrant thyroids will be found to fall into some one of the classifications diagrammatically shown in Figs. 533, 534.

In the case of extruded adenomata it is of some assistance to recognize the so-called pseudo-accessory thyroids shown in Fig. 535 in order that they may be distinguished clinically by their limitation of motion in the outward direction.

Unlike true accessory thyroids, the movement of which is unlimited in all four directions, an extruded adenoma, though it can be moved freely upward, downward, or inward, cannot be moved outward because of the fibrous bands of the capsule still attaching it to the thyroid gland.

Conclusions.—It is of importance to distinguish true accessory thyroids from other tumor masses, if possible.

Lingual goiters require surgical attention, as a rule, only when causing obstruction. One should anticipate the possibility of myxedema following their removal.

Aberrant thyroids are of interest elsewhere only that they may be distinguished from other tumors in the neck requiring removal.

THE HEART IN TOXIC THYROID STATES

BURTON E. HAMILTON

APPRECIATION of the effect of thyroid toxicity on the heart is an important part of the diagnosis, treatment, and prognosis of thyroid disorders: (1) The commoner complaints of patients suffering from thyroid toxicity—forceful rapid heart beat, with undue breathlessness and easy fatiguability on exertion—draw the attention of patient and physician to the heart. (2) These same complaints occur very commonly among patients suffering with neurasthenic states. It is a common diagnostic problem to differentiate heart disease, thyroid toxicity, and neurasthenia. (3) Thyroid disease has a direct effect on the hearts of certain of its victims and can cause disability and death from its effects upon the heart alone, and it is one of few diseases that tend thus to effect the heart seriously.

The most satisfactory conception of the effect of thyroid toxicity on the heart is to regard it as a constant mechanical strain. This stress is present in every case of thyroid toxicity. Commonly the heart rate is distinctly rapid. Even at rest in bed it tends in most definitely toxic cases to beat at a rate above 100, and rates of more than 150 are not uncommon. The highest rate I have counted is 256. An occasional case, however, at rest in bed has a rate of less than 80, and a normal rhythm and rate below 70 has been found with high degrees of thyroid toxicity in rare individuals.

Commonly the heart beat is not only rapid but also forceful. The patient is often conscious of violent heart action and of throbbing blood-vessels, particularly in the neck.

It is the rule in thyrotoxic states, when the heart rhythm is regular, to find the systolic blood-pressure elevated and the diastolic blood-pressure lowered. Though there are occasional

instances when the pulse pressure is one-third or less of the systolic pressure, the pulse pressure tends toward half the systolic pressure or more. There is little evidence to suggest that thyroid toxicity *per se* tends to cause elevation of both systolic and diastolic pressures.

Heart rate and blood-pressure findings are of value among many other points in history and physical examination in the diagnosis of thyroid toxicity, and also in the estimation of the degree of toxicity when the diagnosis is once made.

In the majority of cases the heart tolerates the strain of thyroid toxicity well. In almost two-thirds of all definitely toxic cases one can find no serious heart changes.

The individual hearts in this group do not show *enlargement.* The forcefully beating heart often simulates an enlargement, but careful examination, including teleroentgenograms, shows no evidence of true enlargement in this major group.

Systolic murmurs are common in all thyrotoxic cases, but they are not classifiable, and do not run true to any type.

Diastolic murmurs are not heard except from coincident heart disease.

Though the heart rate in this group is rapid the *rhythm* is normal.

Reduplication of sounds sometimes sufficient to be called "gallop rhythm" is not uncommon. This suggests clinically a myocardial change, but prolonged study of these cases shows that true evidence of myocardial damage, such as heart-blocks, are not associated with these findings except as a rare coincidence.

Heart pain is not uncommon among the thyrotoxic cases. This, however, is not of ill omen, and one never finds cases justifying a diagnosis of a significant "angina pectoris."

The conception of a *direct toxic action* upon the heart causing muscular changes is not appropriate clinically to thyroid disease. It may be that there is such action (most of us instinctively expect to find evidence of heart muscle damage in thyrotoxic cases), but if there is such damage it does not show itself clinically. For example, one does not find among the thyrotoxic

patients heart conditions similar to those occasionally found in diphtheria, where the muscle is often directly damaged. It is much more satisfactory, as stated above, to regard the effect of thyroid toxicity on the heart as a continuous mechanical strain.

Thyroid toxicity picks out certain individuals for definite specific heart damage.

In these individuals it causes first *auricular fibrillation*—an absolutely irregular heart action.

Auricular fibrillation may come on at any time in thyroid toxicity, and may last from a few hours to any length of time. If the attack stops, it may be repeated at any time, and last again for any length of time, but it tends to come on, *first, in transient attacks; second*, the condition *tends to become established.*

Several factors determine whether or not a given case has auricular fibrillation: (1) The *older the case*, the more it is apt to develop; (2) if there is *coincident heart damage*, such as rheumatic heart disease, thyroid toxicity is very apt to cause auricular fibrillation; (3) the more *severe the toxicity*, everything else being equal, the more chance for auricular fibrillation. The onset of an attack is often associated with a clear exacerbation of toxic symptoms, such, for example, as usually occurs to some degree in the forty-eight hours following operative procedures, and improvement in toxic symptoms in individuals with auricular fibrillation is usually accompanied by a return of the heart to normal rhythm.

1. **Influence of Age.**—The following table shows the age incidence of auricular fibrillation in a group of 372 unselected cases, who were definitely and severely toxic:

	Age, years.					
	11–20	21–30	31–40	41–50	51–60	61–70
Number of toxic cases	21	84	105	92	56	14
Percentage with auricular fibrillation	0	13.9	32.4	46.7	64.3	85.7

No case under twenty has had auricular fibrillation, and the percentage, starting with 13.9 per cent. of those from twenty-one to thirty years, increases progressively with each older decade

to 85.7 per cent. of all definitely toxic cases over sixty years of age.

2. **Influence of Coincident Heart Disease.**—An unexpectedly large number of patients with thyroid toxicity have a history of rheumatic fever or chorea or clear signs of mitral stenosis, justifying a diagnosis of a rheumatic heart disease. In the above series of definitely toxic cases about 9 per cent. of the individuals had this rheumatic complication. Probably only 2 per cent. of the communities from which these cases were drawn have a similar rheumatic disease. Two-thirds of all the toxic thyroid cases in the rheumatic group, of all ages, developed auricular fibrillation. The significance, if any, of the clinical association of rheumatic infections in 9 per cent. of the thyrotoxic cases is hidden, but the association is important in the study of the thyroid heart since an appreciable fraction of all cases have this complicating heart disease or potential heart disease.

Though the thyrotoxic cases can be divided into two groups, namely, (1) those with exophthalmic goiter or Graves' disease or *primary hyperthyroidism*, and (2) those with adenoma or adenomatous goiter with toxic symptoms, *secondary hyperthyroidism*, the effect on the heart clinically is the same. Certain individuals of each group are picked out for heart damage according to the same rules, and the heart damage when it occurs is the same in both groups.

Following are two tables showing the members of the group with severe toxic symptoms (tabulated above) separated into these two groups:

Primary Hyperthyroidism

Age, years.	Cases.	Had auricular fibrillation.	Per cent.
Under 20	17	0	0.0
21–30	65	6	9.07
31–40	78	25	32.05
41–50	56	24	42.85
51–60	26	17	65.34
61–70	3	3	100.00

Adenomatous Thyroids with Toxic Symptoms

Age, years.	Cases.	Had auricular fibrillation.	Per cent.
Under 20	2	0	0.0
21–30	14	2	14.28
31–40	15	4	26.66
41–50	30	16	53.33
51–60	26	16	61.53
61–70	9	7	77.77

In the study of the heart in thyroid diseases as well as in the clinical diagnosis of thyroid toxicity it does not help to approach patients as (1) having exophthalmic goiter or (2) having toxic adenoma. It is more practical to regard both groups as suffering from a common *thyroid toxicity*.

A fraction of all thyrotoxic cases with auricular fibrillation show clear evidence of heart failure of the congestive type—"broken compensation." This group is of such clinical importance that it deserves to be considered as a separate problem. I will discuss them in the following article.

With the above brief outline of the effect of thyrotoxic states on the heart in mind, I will discuss the direct clinical handling of the hearts of the cases in the clinic.

All cases, whether clear cut or doubtful, need a careful routine history and physical examination, for in the course of hundreds of individuals with thyroid disease one finds instances of many complicating disorders—tuberculosis, nephritis, diabetes, syphilis, and others rarer. One also finds, as previously stated, complicating heart diseases, but to confine this discussion to the special aspect of history and physical examination, as it is of routine importance to the heart condition in thyrotoxic cases, our procedure is as follows:

Immediately on entrance to the clinic each case is questioned specifically for (1) rheumatic fever, (2) chorea, (3) a history of congestive heart failure (see later), (4) for a history of attacks of disorderly heart action. Certain individuals can definitely tell of seizures of rapid irregular beating of the heart (probably auricular fibrillation) lasting from a few hours to days, and in some cases weeks, with a return to normal rhythm,

though usually rapid rate, after the attacks. Some individuals can tell of such attacks and finally of an attack which has continued to date—a matter of weeks or months or years. Occasionally patients are not aware of disorderly heart action though they have it, and in others it is impossible to distinguish from the description alone whether an attack of violent irregular heart action was a true auricular fibrillation or simply occasional or frequent extrasystoles. (5) All patients are questioned as to whether or not they have been taking medicines, and it is determined, so far as possible, whether they have been using digitalis

One finds at this examination the small but important fraction of cases with complicating rheumatic heart disease; and much rarer cases with cardiovascular syphilis, or with probable congenital cardiovascular anomalies, and a small but very important group subject to attacks of paroxysmal tachycardia, and others with complicating hypertension and heart changes secondary to that. Unless they show true *signs or history of congestive heart failure* (this will be discussed later) all those with a complicating heart disease can be regarded as safe operative risks so far as the heart is concerned. During the last five years we have never been forced to refuse surgical treatment to a thyrotoxic case because of a complicating heart disease.

With the knowledge that thyrotoxic states tend to cause auricular fibrillation in older individuals and in those with previously damaged hearts, and that in each individual the onset of auricular fibrillation is usually associated with strain, or increased toxic symptoms, for example, the increased toxic symptoms following operation, one can separate the cases so far as the heart condition is concerned into the following groups: (1) Those that have never had auricular fibrillation and will probably not have it after operation. (2) Those that have not had auricular fibrillation, but will probably have auricular fibrillation after operation. (3) Those that have had auricular fibrillation and will almost certainly have it after operation. (4) Those that have auricular fibrillation.

The first group need no special care so far as the heart is

concerned. They may be extremely toxic cases and may die of their toxicity, but the heart itself will not fail. The heart is not an essential factor in their condition.

The second group is important to recognize because (1) auricular fibrillation is a distinct burden to the individual, and the surgeon should be forewarned that this additional burden is apt to be placed on this particular patient during the convalescence from operation, and (2) the patient will be watched carefully for the onset of auricular fibrillation by physicians and nurses, so that (3) if the attack occurs proper treatment can be immediately instituted. The attack usually occurs from twelve to forty-eight hours after operation. And its onset is usually coincident with a general exacerbation of toxic symptoms—the so-called "thyroid storms." The attack, as a rule, lasts twenty-four hours or less; occasionally it continues for two or three days, rarely for a number of weeks. In only 1 case out of about 200 thyrotoxic cases that I have seen who had auricular fibrillation for the first time after operation has the auricular fibrillation failed to stop after the relief of thyroid toxicity. An occasional case will have an attack after each of a series of divided stage operations. Others will show an attack after the first operation and not after the following ones. Rarely will an individual who failed to have auricular fibrillation after the first operation develop it after succeeding operations.

The cases in *Group 3*, namely, those that have already had attacks of auricular fibrillation and that have an attack after operation, are important to recognize for the same reasons described above for Group 2, also because some of these Group 3 individuals deserve digitalization in anticipation of a certain attack of auricular fibrillation. Such attacks may start in this group of cases at any time. Several times in our experience an attack was started during an operation. The heart behaves much better in such individuals during an attack if they are properly digitalized. (See later.) Every one of our Group 3 cases has *returned permanently to normal rhythm after relief of their thyroid toxicity*. Some of the cases in this group are very striking. For example, a woman sixty years of age had been

subject to attacks for twelve years. These grew so frequent that they occurred twice a week during the year before operation. The attacks tended to last about nine hours. During an attack the patient was very uncomfortable and developed true signs of congestive heart failure, which cleared within the twenty-four hours after the attack when the patient was at rest in bed. The patient was disabled completely and practically bedridden for two years before she came for operation. Following the final operation she failed to have an attack, and she has not had one during three years that we have been able to follow her since relief of her thyroid toxicity. This story in essentials can be repeated for many individuals. There is in the community another small group of individuals who are subject to repeated violent attacks of auricular fibrillation and have no underlying hyperthyroidism. These cases are very troublesome. They tend to continue to have attacks whatever one does for them medically. But the individuals who have an underlying thyroid toxicity that I have seen, and they form a considerable group, have been immediately and permanently relieved of their attacks when the thyroid toxicity was cured by surgery, and this relief has been without the aid of drugs, except for digitalization during the attacks. We have also had 3 striking cases of adenomatous goiters, but no clear clinical evidence of toxicity, and with normal basal metabolism readings, who were subject to repeated paroxysms of auricular fibrillation for a long period before reporting to us. Though we believed that there could be no association between the thyroid disease and the auricular fibrillation since evidence of toxicity was lacking, the adenomatous thyroids were treated surgically, and each one of these cases has as the result of this been freed from attacks of auricular fibrillation. It is probable that these cases did have toxicity associated with the adenomatous thyroid, even though the toxicity could not be measured by ordinary clinical procedures and basal metabolism estimations.

The *fourth group*, cases that have established auricular fibrillation, in more than one-third of the instances return

permanently to normal rhythm within three months of relief of thyroid toxicity by surgery. These cases have had the auricular fibrillation for months and, in some instances, for years.[1] All of the above group can be considered "safe operative risks" so far as the heart is concerned, if the burden of auricular fibrillation is taken into account, providing the patients do not show, or have not recently had, congestive heart failure. (See later.)

The **diagnosis** of auricular fibrillation is usually an easy matter. It depends on an appreciation of a continuously disorderly heart action. At times it may simulate orderly heart action so closely that it may be overlooked. Rarely the normal fluctuations of normal rhythm occurring with excitement, activity, respiration, etc., are aggravated in some of the thyroid cases to such a degree that an auricular fibrillation is strongly suggested, but usually the condition is recognized at once by any observer who knows that there is such a thing as auricular fibrillation and is on the watch for it. Surgeons, house officers, and nurses readily recognize nearly all cases of auricular fibrillation. It happens more than once each week that a nurse will call me at night and report that some patient has developed an auricular fibrillation, and the report is almost always correct. Electrocardiograms are occasionally taken, and they, of course, will diagnose the condition easily, but no one dealing with thyroid patients should be unable to recognize this condition of the heart-beat on direct auscultation of the heart except in rarest cases. And, in fact, all physicians and surgeons should be aware of auricular fibrillation and train themselves to recognize it.

Treatment of Auricular Fibrillation.—It has been the rule

[1] It is possible that some of these cases with established auricular fibrillation persisting after relief of thyroid toxicity would justify quinidin therapy, but because these individuals that I have seen have done well under digitalization, and because they have been without exception very sick individuals before surgical treatment of the thyroid disease and have later shown a rather astonishing return to full ability, it has not seemed wise to me to venture the risk, and it is a risk, of quinidin administration. I can see no clear place for quinidin therapy in hyperthyroidism with auricular fibrillation.

in this clinic for the last five years never to give digitalis to cases that do not have auricular fibrillation. The only exception to this rule is the occasional case that is certain to have an attack of auricular fibrillation, though not actually in an attack at the time of entrance to the clinic. All the cases with auricular fibrillation are digitalized as soon as the diagnosis of auricular fibrillation is made.

At the risk of repeating common knowledge I will discuss the use of digitalis in thyroidism. It was until recently fairly common in medical practice everywhere for cases of rapid heart rate, but regular normal rhythm, and, of course, this will include the cases with thyroid toxicity without auricular fibrillation, to be given digitalis. It was also common for patients in general who were considered poor risks to be given digitalis in order to strengthen the heart before operation.

A growing knowledge of the behavior of hearts and of the effect of digitalis has tended to change this. It is now rather generally believed that it is not helpful to give digitalis to people with normal heart rhythms. When the rhythm is normal it is difficult to lower even a rapid rate with digitalis. And it is known that it is very doubtful if one can lower the heart rate appreciably in hyperthyroidism with digitalis providing the heart *rhythm* is normal. Digitalis is not in one sense a heart stimulant. It is, more properly, a heart poison. In itself it may cause a disorder of the heart-beat in certain individuals. And though there is perhaps little definite evidence to justify this attitude, men of sound clinical judgment and special training in diseases of the heart usually feel that it is wise not to give the heart the possible added burden of a digitalis-poisoning unless there is some definite benefit to be gained by it. Equally sound clinical judgment based on numberless observations holds that where auricular fibrillation is present the heart poison—digitalis—is helpful. In auricular fibrillation the ventricles beat in response to haphazard, rapidly, and irregularly repeated impulses from the auricles. Digitalis in poisoning the heart muscle renders it less irritable to these unduly rapid and disorderly stimuli, blocks many of them, and as a result there is a

slower and more orderly ventricular action. It is believed that the heart can behave more effectively in auricular fibrillation when proper doses of the drug are given. This has been shown by a multitude of direct and indirect observations readily accessible to anyone interested in confirming this almost universally recognized fact. Furthermore, digitalis has a tendency to abolish auricular fibrillation and cause a return to normal rhythm. We have given it in several hundred cases of hyperthyroidism with auricular fibrillation—about two-thirds of these cases had "primary hyperthyroidism," Graves' disease, and one-third had "secondary hyperthyroidism," toxic adenoma.

It is a very common thing for me to see cases with auricular fibrillation with a heart rate well above 120, sometimes as high as 200, and occasionally higher, drop to almost one-half this rate under proper doses of digitalis, and with the drop there is usually a reduction in the number of apparently ineffectual beats, those which fail to cause an appreciable pulse wave at the wrist. There is also often a distinct subjective improvement with the relief of the tumultuous heart-beat. One can count on a definite reduction in heart rate in *auricular fibrillation* and thyrotoxic states under proper doses of digitalis. Occasionally the heart rate will drop from nearly 200 to below 80. I have never seen a death or serious symptoms develop which could be reasonably attributed to the influence of digitalis in this clinic. A number of cases who have died have had auricular fibrillation for periods of hours or years before death, and have had digitalis as long as they had auricular fibrillation and were under our care, but I can find no case whose death was apparently hastened by digitalis, on careful study of the records of these patients, individuals whose clinical course I have personally watched.

One is discarding a valuable, trustworthy, and easily available aid if one discards the use of digitalis in auricular fibrillation in thyrotoxic individuals.

The method of giving digitalis that we use is based on prolonged clinical trial, carefully observed. An individual who has had no digitalis or digitalis-like drugs for two weeks can

stand between 18 and 24 grains of a standardized powdered leaf at a single dose. This dry form is used because the liquid forms are believed to be less stable, and if uniform dosage is desired the liquid forms would require very frequent standardization. There is no evident advantage in the liquid forms to offset this difficulty. Usually 18 rather than 24 grains are given. Sometimes, where 18 grains have been given, 6 more grains are given after eight hours. After this dose none is given for twenty-four hours, and then 3 grains daily usually serves to keep the patient digitalized. Often this dose has to be reduced to 2 grains daily after a few days. Sometimes an occasional day without digitalis is required. The clinical symptoms suggesting too much digitalis are as follows: (1) Coupling of the beats, (2) vomiting, (3) nausea, (4) loss of appetite without marked nausea, and with a sense of prostration associated with a slow heart rate, and (5) rarely, a diarrhea without the other signs.

It is a very rare thing to have difficulty in satisfactorily digitalizing thyroid patients with auricular fibrillation. Cases that "cannot take digitalis" are unknown to my experience with thyroidism. A very rare case has taken larger amounts than the above before the effect of the drug was noted. Though patients vary somewhat in the amount that they require to keep them digitalized, I do not recall a case in this group that has been satisfactorily digitalized on much smaller doses than the above. Frequently it is necessary to give digitalis to patients after operation when they are vomiting. It is usually possible to digitalize them by mouth administration in spite of vomiting, which is often present after operation at just the time when auricular fibrillation starts. Where the drug is vomited at once repeatedly, the same dosage has been given in a few cases by rectum. The powdered leaf has been ground up and mixed in 2 ounces of water and given as an enema. I have very rarely used subcutaneous or intramuscular preparations. The cases that may develop an attack of auricular fibrillation during operation may be recognized before operation, and those who have chronic auricular fibrillation (which may be overlooked) can nearly always be found if searched for, and digitalized carefully

before operation. Fortunately, the cases that develop auricular fibrillation only after operation tolerate the auricular fibrillation well. They have not except in rare instances shown signs of congestive failure at this time for the first time, so that the indication for immediate digitalization at all costs parietally is not strong. Fortunately, again, digitalis action can be appreciated clinically four to six hours after administration of proper doses of the drug by mouth. Almost always satisfactory results can be obtained within eight hours, providing a proper dose has been given. Only very rarely are the clinical results delayed for more than eight hours when the full dose has been given.

Unfortunately, many patients have been taking varying amounts of digitalis of unknown strength before reporting to the clinic. In these individuals it is important to feel one's way to avoid unpleasant symptoms. The 18 grains may be divided into three doses of 6 grains eight or twelve hours apart, and the patients examined before each dose is given, and studied for signs of physiologic action as well as for signs of overdigitalization. The proper dose is usually best determined by the clinical response of the patient—a satisfactory slowing of the heart rate. Though electrocardiograms are taken whenever desired as a help to the study of digitalis therapy in these cases, with increasing experience the number of cases where cardiograms are desired is small.

It is distinctly advisable not to cause vomiting in these cases. Overdigitalization may lead to three whole days of nausea, after the drug is stopped, with refusal to take food, or vomiting of all food if taken, and the attendant necessity of supplying the patient with fluids and some nourishment parietally. It is very rare if proper precautions, as above, are taken, that one cannot digitalize any patient within twenty-four hours to a satisfactory clinical degree and still avoid unpleasant symptoms. Satisfactory digitalization within eight hours is the rule. It has been more than two years since severe nausea, as described above, has followed digitalization in our clinic.

It is a matter of clinical judgment whether or not one should digitalize all the cases expected to have auricular fibrillation after operation. Some of the expected attacks do not arrive. When they do arrive there is still plenty of time to digitalize the patient. In my opinion it is wiser, except where auricular fibrillation is present or where one is morally certain that it will occur, to avoid the use of digitalis. The thyrotoxic cases that do not have auricular fibrillation do not show heart failure.

The space taken to discuss digitalis is due to the difficulty of discussing it adequately in a few words. *Digitalis is not the most important thing in the treatment of thyroid hearts*, though it is of definite value when properly used. The important things are: (1) *Recognition of coincident heart disease*, (2) *anticipation of auricular fibrillation*, (3) *not to be afraid of hearts, however damaged, if they are doing their work*, and (4) *to recognize the actual significant signs of a failing heart.*

HEART FAILURE (CONGESTIVE) ASSOCIATED WITH THYROID TOXICITY

BURTON E. HAMILTON

DURING the last four years I have found in this clinic a distinct group of cases who showed clear evidence of congestive heart failure associated with thyroid toxicity.

Congestive heart failure is a definite clinical finding. It is usually easy to recognize, but at times in certain individuals it is readily confused with respiratory tract diseases and with abdominal disorders, even with acute surgical abdominal conditions. By congestive heart failure is meant simply evidence that the heart is failing to remove the blood from the veins as fast as they fill. The resulting *venous congestion* shows itself by the following signs: (*a*) From congestion in the pulmonary veins, orthopnea; cough, which may be productive and may yield bloody sputum; râles, usually found most numerously, and often only found at the bases of the lungs posteriorly; diminished vital capacity. (*b*) From congestion in the systemic veins, enlargement of the liver, and secondary to this enlargement there may be tenderness, pain, and sometimes jaundice; overfulness of the neck veins may be demonstrable, but it is a difficult sign to be sure of.

There may also be general demonstrable edema, but this is not an essential part of the picture, and when present is best considered as a late result of the venous congestion.

The two venous systems are usually simultaneously involved. Only occasional cases show the lung congestion without demonstrable liver engorgement, and rare cases have marked liver engorgement without signs in the lungs.

Congestive failure may come on in a moment or slowly, and may vary greatly in degree. Occasionally patients may be

up and about, and at least attempting to work, with gross congestive failure present. The symptoms tend to be markedly aggravated by exertion.

"Breathelessness, rapid heart, edema" are a triad often thought of as signs of cardiac decompensation, but by themselves they are not evidence of a failing heart. For example, rapid heart and breathlessness are almost always present to some degree in thyroid toxicity, and yet congestive heart failure is present only in occasional thyrotoxic cases.

One can divide all cases that are breathless and have rapid heart rates, and this includes nearly all individuals that are severely sick into two groups: (1) A small group whose veins are congested because the heart is not doing its work. (2) A large group whose veins are not congested, and one can be sure that the heart is doing its work.

The importance of this differentiation in treatment of acutely sick patients is evident. For example, in emergency treatment, transfusion or intravenous saline solutions to improve a failing circulation are contraindicated if there is venous congestion; bleeding is sometimes directly indicated. The exact opposite is true when, as is more common in dangerously sick patients, the veins appear to be relatively empty and the blood is feebly returned to the heart from a failure of the vascular part of the circulatory apparatus, not of the heart itself.

True congestive heart failure is found in only a small group of individuals. Patients with damaged hearts, for example, with rheumatic heart disease, cardiovascular syphilis, or in the degenerative changes associated with arteriosclerosis or with this plus a hypertension, often show congestive heart failure from a progress of their heart disease, or when under some unusual coincident strain, such as an illness. But there are few mechanical strains or diseases that by themselves tend to cause congestive heart failure in people with relatively sound hearts.

For the above reasons it is clear that patients with congestive heart failure of whatever underlying cause, and with whatever complicating disease, deserve to be considered as a special group. And it is, therefore, of great interest to note

that in thyrotoxic patients in a thyroid clinic true congestive heart failure is found in a small but distinctly important group of individuals. I have found it in a small number of the cases subject to transient attacks of auricular fibrillation, and in a larger number of the actually smaller group that have persistent auricular fibrillation. (See previous article.)

The group that shows congestive failure in association with thyroid toxicity contains, as is to be expected, a small number suffering clearly with severe *coincident* heart damage, but the others (more numerous) are individuals that so far as can be told would not be likely to develop heart failure under any ordinary strain, and the strain of thyroid toxicity can be considered the prime factor in their heart failure.

Diagnosis of Thyroid Toxicity Underlying Congestive Heart Failure.—The diagnosis of thyroid toxicity, as has been discussed, is often very difficult. It is particularly difficult when there is also present congestive heart failure dominating the clinical picture and obscuring the underlying cause. The majority of the cases that I have studied tell a history of prolonged disability from congestive heart failure before thyroid toxicity was suggested by their condition or could be thought of. Altogether they present a picture not commonly seen in heart diseases, namely, they have gross signs of congestive failure from which they cannot be relieved by prolonged rest in bed, other means of sparing the heart, and digitalization, and yet (in most instances) they manage to live on for months or years. An occasional case in this group has had a period of disability from congestive heart failure with relief for a long period and return of symptoms, a haphazard onset and offset of their failure which can best be explained by fluctuations in thyroid toxicity. A few have had sudden brief attacks of failure repeated many times, which were clearly in association with paroxysmal attacks of auricular fibrillation. This in itself suggests the possibility of thyroid toxicity.

The following table is designed to show the incidence of some of the commoner thyrotoxic symptoms, and the prominent general symptoms in thyrotoxic patients with congestive

heart failure. It is a summary of the signs presented by 50 such cases whose diagnosis was proved by all available means.

	Per cent.
Heart:	
Enlargement	100
No diastolic murmurs	90
Auricular fibrillation	96
Rate above 100 (after rest and digitalization)	72
Exophthalmos (definite)	16
Goiter:	
No	16
Doubtful or slight	74
Definite	10
Edema	94
Emaciation	100
Pigmentation (definite)	72
Mental apathy (as a prominent symptom versus overactivation which is rare in this group)	56
Sex: Women 88 per cent. Men	12
Age: Average 50 years. Over 40 years	80

One can see from the above that one cannot find any set of symptoms which is of great diagnostic value and which is infallibly present in such cases. Every important finding suggesting thyroid toxicity is inconstant to some degree, but one can construct from the study of these cases a *composite picture.* The typical patient with congestive heart failure caused by thyrotoxic states would be a *woman* close to *fifty years* of age She would be *emaciated,* would weigh about 100 pounds (or tell a clear story of having lost unaccountably a large amount of weight). She would not be mentally overactivated as is usual in thyroid toxicity, but would tend to be *apathetic,* willing to lie in bed and unable to make an effort to exert herself mentally as well as physically. Her *heart* would be *enlarged* and she would have an *absolutely disorderly rhythm.* The *rate* would tend to be rapid and only with difficulty, in spite of full doses of digitalis, could it be kept below 100. She would have *no diastolic murmurs* or other evidence of complicating heart disease. She would have the *signs* of *congestive heart failure* and gross edema, and this *failure would be particularly resistant to medical treatment.* It would be apt to persist in spite of prolonged rest in

bed and digitalization, or if yielding slowly to these measures would return promptly on exertion. She would probably have *none of the eye signs* of thyroid toxicity and *no goiter*.

The individual cases tend to run very true to this type. Though they vary from it they tend to vary in only one or two or, at the most, three points.

Basal metabolism estimations taken to confirm a thyroid toxicity, when this is suspected in such an individual, are very difficult to interpret. People with congestive heart failure tend to have elevated basal metabolism readings even when they have no underlying thyroid toxicity. I have seen such cases give readings as high as plus 60, and often higher than plus 50. And more than half of the cases with thyroid toxicity underlying their heart failure have had readings of less than plus 50 on the day before operation. It is probable that a normal basal metabolism reading should serve to exclude the possibility of an underlying hyperthyroidism in a patient with present or recent congestive heart failure, but except for this negative value basal metabolism readings must be considered as of very slight aid in confirmation of the diagnosis.

To be sure, a patient with congestive heart failure and *no* underlying thyroid toxicity and an elevated basal metabolism reading is almost certain to have normal basal metabolism readings after complete relief of the congestive heart failure, but the majority of cases of heart failure with underlying thyroid toxicity cannot be made rid of their heart failure except by surgical treatment, and the basal metabolism readings must be interpreted as taken in the presence of a condition which by itself tends to elevate them to an uncertain degree.

The difficulties of diagnosis in this group of individuals and the case histories of many that I have seen suggest that there must be *individuals scattered through the community suffering from congestive heart failure and diagnosed and treated as having uncomplicated heart disease when they have actually an underlying thyroid toxicity, which is the real cause of their failure.* We should (1) remember thyroid toxicity as a rare cause of congestive heart failure; (2) bear in mind the typical

case picture; (3) recognize the difficulty of diagnosis and endeavor to confirm it by all the aid that prolonged clinical experience can bring to bear on each individual case. Most diagnostic errors that I have seen in this group of cases have been failures to recognize thyroid toxicity rather than to suspect it wrongly. (The latter error, however, is easy to make. One is tormented by seeing signs suggesting thyroid toxicity in too many chronic cardiac cases after the typical thyrocardiac is in mind.)

There would be little incentive, perhaps, to search for a rare thyroid toxicity as a possible cause of congestive heart failure were there no satisfactory treatment. The response of these individuals to surgical treatment is truly astonishing.

I will describe briefly the individual case that served best to call attention to the diagnostic difficulties and to the possibilities of surgical treatment of thyroid toxicity causing congestive heart failure, though she was not actually the first case of this sort that was operated in the clinic.

Mrs. A., aged fifty-six, childless. Unimportant past history; no rheumatic infections; no lues. Present illness (on reporting to the clinic): Increasingly disabled by breathlessness on exertion for one and a half years. During the last six months she had been in bed nearly all the time. She had not been able to lie down for at least six months because of smothering sensation; she had had persistent gross edema of her legs for this length of time. She had been constantly under the care of a very able internist. At the time of our examination her *eyes*, nose, throat, and neck were normal except for overfulness of the superficial neck veins. Her thyroid gland could not be palpated. Hurried respiration, unable to lie down; râles at both bases posteriorly. She showed a general brownish pigmentation of the skin, more marked on the eyelids. Mentally she was alert and gave the impression of being overactivated. Her heart was grossly enlarged, moving her chest wall with each beat; absolutely irregular, rate about 130; systolic murmur heard everywhere over the precordium; no diastolic murmurs. Abdomen was prominent and showed evidence of

ascites. Her liver edge could be made out with difficulty, halfway between the costal margin and the umbilicus. She had gross edema of her legs, extending up the belly wall and partly up the chest wall. In spite of this edema she appeared emaciated. Her basal metabolism was found to range between plus 50 and plus 60.

She was rejected as a surgical possibility for two reasons: First, it was believed that she could not possibly survive operation, and second, there was grave doubt felt by all concerned as to whether or not she had hyperthyroidism.

She remained under our care, and in spite of rest in bed in hospital with special nursing, thorough digitalization, and all the aid of diet, medication, etc., that could be thought of to improve her condition, she remained absolutely stationary.

Because she would not die and could not get well Dr. Lahey finally did a pole ligation under local anesthesia as a more or less desperate measure. She tolerated the ligation well, and for the first time showed a slight improvement. Subtotal thyroidectomy was completed six weeks later. Following this she lost all signs of congestive failure. Three months later she was up and about doing housework for herself and her husband, and after a few months took up music teaching in addition. She has remained without complaints and with no signs of heart failure. It is now two and a half years since completion of her operation.

Since this instructive case every similar case that I have seen has had surgical treatment. There have been, however, 8 individuals with congestive heart failure that in all probability had underlying thyroid toxicity, who were in such extreme condition that in spite of all emergency treatment (including in the last 2 cases large doses of Lugol's solution and in several of the others Lugol's was given to some extent) died within a few hours or days from the time they were seen. Roughly, two-thirds of these cases operated still showed congestive failure at the time of operation.

Two cases have died in connection with surgery. One died suddenly and unexpectedly twelve hours after a subtotal thy-

roidectomy (three years ago). She had previously survived two operations, and was apparently in better condition for the third than for the previous two. The other died suddenly and unexpectedly during induction of anesthesia before her second operation could be started (six weeks ago). Autopsy failed to show a clear cause of death. All the others have survived surgical procedures.

It is not suggested by this that one can operate safely out of hand on people with congestive heart failure and underlying thyroid toxicity. These successfully operated cases have been very carefully prepared, and so far as was possible the most opportune moment chosen for operation. Their individual problems have been studied carefully, and preparation for operation, type of anesthesia, operative procedure, and medical and surgical after-care have been carried out with all the ingenuity that the individual physicians, surgeons, and nurses concerned could afford to each individual case, as though it were the most important possible case. They have had rest in bed for a period of at least three weeks to a maximum of six months before the first operative procedure. Actually they have tolerated operation surprisingly well and have required no greater number of operative stages than the general run of thyrotoxic cases without congestive heart failure.

Since the first of this year 16 individuals of this group have been operated. One died (quoted above). Another, who had been disabled by her congestive heart failure for several months before operation in spite of rest in bed, was operated successfully in two stages, went home and resumed slight activity, had a sudden hemiplegia, probably embolic in origin. It is too soon to quote data as to return of full ability in this group of cases, though many of them have already returned to active duty.

Thirty-nine cases were operated before January 1, 1924. Two cannot be traced. One died at operation (quoted above). *All the others were completely relieved of their congestive failure.*

Five have since died.

Case I.—A man aged sixty-one, who had been completely disabled for three years, was operated on when in heart failure, lost all signs of heart failure, and was apparently well for several months. Against advice he began heavy manual labor on a farm, had a return of his congestive heart failure, and died four months after operation. Necropsy showed marked arteriosclerosis with the heart and kidneys involved.

Case II.—A woman, who had coincident rheumatic heart disease with complete disability from congestive heart failure for one year, and was operated on when in heart failure, had complete relief for a few months, but resumed violent activity against advice. Heart failure returned, and she died six months after operation.

Case III.—A woman aged forty-eight, bedridden with severe heart failure for five months before operation, was operated on in heart failure and completely relieved. She resumed work as a secretary, but died, after a brief illness, of lobar pneumonia, eight months after operation.

Case IV.—A woman aged sixty-one, increasingly disabled by congestive heart failure for at least two years before her operation, was afterward completely relieved and able to maintain full activity; three years later she was operated on for a pelvic tumor, and died of general peritonitis.

Case V.—A woman aged thirty-five, with chronic rheumatic heart disease, mitral stenosis, aortic regurgitation, and auricular fibrillation, had been disabled with continuous congestive heart failure for two years. She had a moderate goiter and prominent eyes, was nervous, had a basal metabolism of plus 24—the thyroid toxicity had apparently lasted for two years. She was operated safely, relieved of her congestive failure, and able to be up and about with restrictions as to climbing, carrying, etc. She died suddenly and unexpectedly two years after operation.

The others, 31, are maintaining full activity. They have at present gone an average of about two years with full activity, and they had an average complete disability of twenty months before operation. Their individual case histories would be instructive and most satisfying to detail, but the first case quoted in this article serves to illustrate them.

In dealing with patients of all sorts suffering with congestive heart failure one can find no experience to compare with this group of individuals with an underlying thyroid toxicity treated surgically. They have as a group been relieved safely by surgical measures from prolonged apparently hopeless disability with gross congestive heart failure, and have resumed full activity, which they can sustain.

LARYNGEAL PARALYSES AND SURGERY OF THE THYROID

D. Crosby Greene

The laryngeal changes associated with goiter and surgery of the thyroid are dependent upon lesions of the recurrent laryngeal nerve. Partial or complete paralysis of one or both vocal cords may result from pressure of an enlarged thyroid upon the nerve, or from an injury to the nerve during operation, or from the pressure of a hematoma or inflammatory exudate upon the nerve secondary to operation.

The motor innervation of the intrinsic muscles of the larynx is partly derived from the superior laryngeal nerve as well, but since this nerve lies well above the region of the thyroid gland, it is not subject to pressure from enlargements or tumors of the gland, or to trauma incident to surgical procedures.

The only intrinsic laryngeal muscles supplied by the superior laryngeal nerves are the cricothyroid muscles, whose function is to tense the cords, and the arytenoideus muscle, whose function is to complete the approximation of the cords in phonation. The latter muscle is supplied by fibers from both the superior and the recurrent nerves.

The rest of the motor nerve supply of the larynx is derived entirely from the recurrent nerve, whose fibers are distributed to the muscles which govern abduction, adduction, and relaxation of the cords. Any trauma or lesion of the recurrent nerve results in impairment of function of these muscles, and is shown on laryngoscopic examination by characteristic positions assumed by the vocal cords. For practical purposes we recognize three cardinal positions of the cords in reference to opening and closure of the glottis, namely, abduction, the position assumed during ordinary respiration, the cords being widely separated

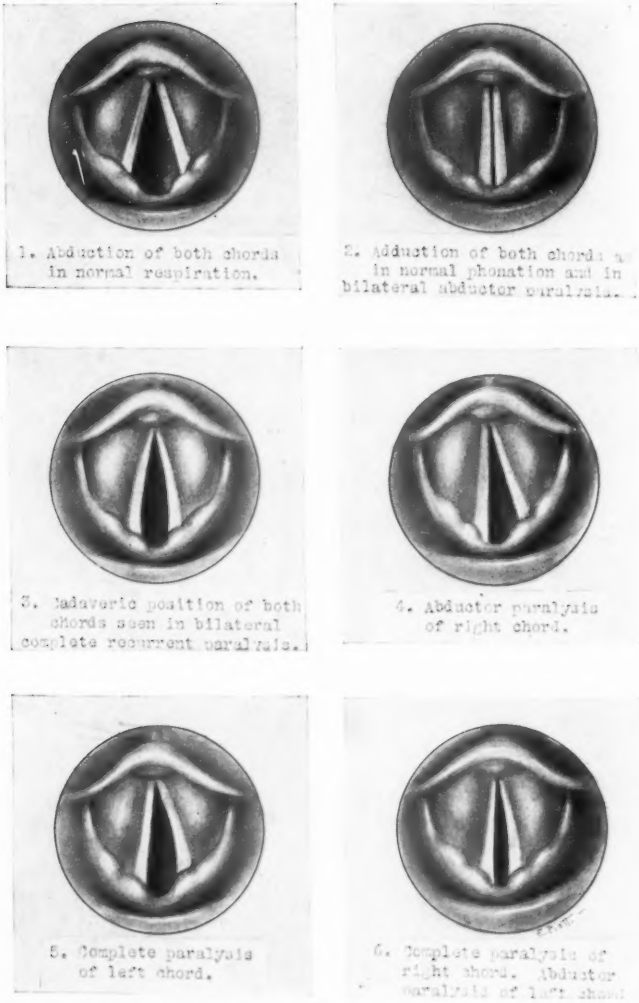
1. Abduction of both chords in normal respiration.
2. Adduction of both chords in normal phonation and in bilateral abductor paralysis.
3. Cadaveric position of both chords seen in bilateral complete recurrent paralysis.
4. Abductor paralysis of right chord.
5. Complete paralysis of left chord.
6. Complete paralysis of right chord. Abductor paralysis of left chord.

Fig. 536.—Diagrammatic representation of positions taken by the vocal cords under normal conditions and in paralyses of the recurrent nerve.

(Fig. 536, 1); adduction, the position assumed during phonation, the cords being approximated in midline (Fig. 536, 2), and the cadaveric position, taken when there is complete paralysis of the cords, in which they remain fixed about midway between abduction and adduction (Fig. 536, 3).

The vocal and respiratory symptoms accompanying complete or partial paralysis of the recurrent nerves correspond with the degree of paralysis affecting one or both cords. Thus, in bilateral complete paralysis, both cords being in the cadaveric position, the voice is completely lost, but there is no dyspnea. In bilateral abductor paralysis, the cords being fixed in approximation, the voice, although changed, is not lost, but dyspnea is pronounced. In unilateral complete paralysis (Fig. 536, 5) the voice is definitely changed and weakened, but, as a rule, there is not complete aphonia, and there is no dyspnea. In unilateral abductor paralysis (Fig. 536, 4) the voice is fairly strong, though somewhat altered in quality, and there is some dyspnea on exertion. The combination of complete paralysis of one cord with partial paralysis of the other (Fig. 536, 6) results in loss of voice and dyspnea on exertion.

All of these various forms and combinations of paralysis have been observed in cases of goiter before or after operative procedures. The recurrent nerve appears to be extremely vulnerable, and a slight degree of trauma may result in impairment or loss of function of the corresponding cord.

It has been the generally accepted view of laryngologists since the days of Semon that fixation of the cords in the midline represents a lesion of only those fibers of the recurrent nerve which supply the abductor muscles; in other words, that this condition represents a partial paralysis of the nerve. Nevertheless, it is, in effect, a complete paralysis, since the midline position is maintained constantly. In goiter cases this form of paralysis is the type which is most frequently met with. It may occur as the result of pressure by the gland before operation, or as the result of trauma to the nerve during operation, or as the result of pressure by a hematoma, or an inflammatory exudate, or a cicatricial contraction after operation. Bilateral

abductor paralysis is dangerous, since the occurrence of even a moderate degree of inflammatory swelling in the region of the cords may be sufficient to cause urgent dyspnea and death. When it is present tracheotomy should be done as a safety measure.

In regard to the prognosis: In cases of paralysis due to pressure from the goiter we have never seen a recovery of function of the cords, even after removal of the tumor.

In cases of paralysis due to operative procedures recovery of function may take place. We have seen a case of bilateral abductor paralysis operated in another clinic in which the cords remained immobile for over six months, and then gradually recovered complete function. In most of the cases in which recovery takes place the time is much shorter, and beginning movement of the cords may be observed in a few weeks.

The effect of trauma to the nerve during operation appears immediately in alteration or loss of voice. In some cases, however, the signs of paralysis come on after operation, and in these pressure from hematoma is the apparent cause. In our experience the prognosis is better in the latter class of cases, although not invariably good.

In the majority of cases laryngeal paralyses resulting from thyroid operations are permanent. As we have observed, the one type which causes urgent symptoms is bilateral abductor paralysis. When this is present immediate tracheotomy is indicated, and later, if the paralysis persists, some measure for permanently relieving the stenosis at the glottis.

TREATMENT OF THE EYES IN EXOPHTHALMIC GOITER

E. B. Dunphy

In exophthlamic goiter the eyes demand the most careful supervision next to the heart and circulation. Since corneal ulceration due to lagophthalmos, when once started, often rages unchecked and leads to partial or complete blindness, it behooves the physician in charge of a case of exophthalmic goiter to treat the eyes prophylactically and not wait until corneal ulceration has developed.

The damage to the cornea is not due entirely to the exposure from the exophthalmos, but in part to a spasm of the levator palpebræ muscle which keeps the upper lid retracted and causes pressure on the anterior segment of the eye (Dalrymple's sign). There is also a diminished tendency to perform the act of winking (Stellwag's sign), thereby increasing the liability to corneal desiccation.

From the onset of the exophthalmos the eyes must be observed most carefully. If the patient can close the lids together the cornea is, of course, protected. It must not be assumed, however, that the cornea is protected during sleep just because the lids can be approximated by a voluntary effort during the daytime. It is important to have the nurse or some member of the family observe the patient during sleep and report if any part of the sclera or cornea shows between the lids. Fortunately the eye rolls up beneath the upper lid during sleep so that the cornea is usually protected even if the lids are not absolutely approximated.

In such cases, where the sclera alone shows, a liberal supply of vaselin should be instilled in the culdesac upon retiring. This will suffice to protect the eye during the night from conjunctival irritation.

In the daytime the winking function, although diminished, is usually sufficient to keep the cornea moistened. If the absence of winking is marked, it is best to instill vaselin three times a day, for although the cornea may not ulcerate, there is a tendency toward desiccation which, if not taken care of, probably weakens the cornea and makes it more prone to ulceration later on.

In cases where the lowest part of the cornea is visible during sleep, it is not sufficient simply to rely on vaselin. The lids must be approximated. It is perfectly useless to attempt to accomplish this by the conventional method of bandaging the eyes, as the palpebral fissure always gaps beneath it. The lids should be strapped firmly together with adhesive tape, and if care is taken in doing this, the cornea will be well protected for the night. The method I have found to be the most efficacious is as follows:

The culdesacs are first irrigated with weak boric acid solution and all bits of secretion removed from the lid margins. The lids are then thoroughly dried with cotton. A strip of adhesive tape 3 inches long and $\frac{3}{4}$ inch wide is split lengthwise half its length. Each of the two split ends is applied to the upper lid (as in diagram, Fig. 537). A second piece, $\frac{1}{2}$ inch long and $\frac{1}{2}$ inch wide, is applied, one end to the lower lid, the other end being brought up between the Y formed by piece A. When these are firmly in place, the patient closes his eyes shut and the lower part of A is pulled down and applied to the cheek at the same time the upper part of B is pulled up and applied to the upper lid. These are held in place for a few moments to be sure they stick firmly to the skin. Both eyes should be done and the patient cautioned not to try to open the eyes. If attention is paid to detail, this strapping will hold in cases not too far advanced. The strips should be removed next day and powder applied to the skin to prevent irritation. On account of the increased tonus of the levator no strapping will be efficient more than a day. The secretions of the eye tend to loosen things up, so the operation must be repeated daily.

If ulceration of the cornea has already developed, radical

measures must be resorted to for preservation of the eyes. The palpebral fissure should be closed by sutures. The lid margins just behind the cilia are denuded for about 10 mm. and the two raw surfaces approximated with a couple of fine silk sutures. The rest of the palpebral fissure should be closed by more sutures without denudation of the lid margins. Care must be taken that the needle is not introduced through the tarsus,

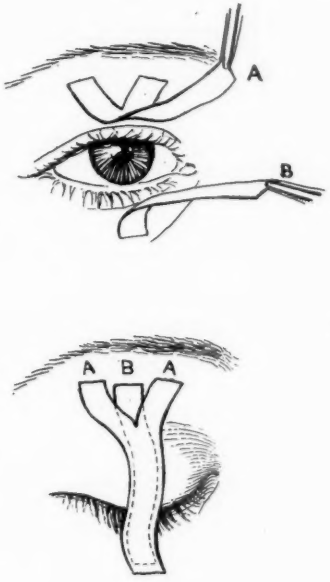

Fig. 537

which would cause disfiguring notches in the lid margins later on. It is also advisable to have the sutures go through the lid as close to the edges as possible, otherwise the lashes have a tendency to roll in. A canthotomy at the external canthus will give greater relaxation of the lids for the closure. Atropin 1 per cent. solution should be instilled at the outer canthus twice a day. Cold compresses are indicated if there is much edema of the lids following the operation. In applying these

it must not be forgotten that we are, after all, treating the cornea, and the nutrition of that part must not be impaired by too cold or by too long application of the compress. Ten minutes every three hours is enough. The sutures should be left in place until they pull out.

In moderately severe cases of exophthalmos of long standing a tarsorrhaphy can be done at the outer canthus to shorten the palpebral fissure and allow the patient to close the lids fairly well himself. This does away with strappings and sutures, which are only temporary measures and cannot be continued indefinitely.

In cases of rapidly increasing exophthalmos where other methods fail, success is sometimes obtained by removing a quantity of edematous fat from the orbit through the lower fornix. Failure is more often the case in this method, as the exophthalmos continues unabated in spite of the treatment.

In the very severe cases where luxation of the eyeball threatens, decompression of the orbit by removal of the temporal wall is justifiable. There may be disagreeable after-effects due to abnormal function of the external rectus muscle.

It is advisable in doing a tarsorrhaphy to cut the temporal fixation fibers and lateral ligament. This will lessen the pressure on the anterior segment of the globe. If the relaxation of the lids is not sufficient, a vertical tarsotomy at the medial lid angle can be done. In spite of all this the retracting tendency of the levator may still be dangerous. The tarsal surface of the upper lid may be enlarged and an attempt made to abolish the action of the levator on the lid margin by severing conjunctiva, tarsus, and orbicularis parallel to the inner edge and about 3 mm. above it from medial to lateral margin.

DIFFICULTIES OF DIAGNOSIS OF TOXIC SYMPTOMS REFERABLE TO THE THYROID GLAND

BURTON E. HAMILTON

PATIENTS are suspected of thyroid disease and apply to a thyroid clinic (1) because they have or think they have a goiter, or (2) show symptoms suggesting thyroid toxicity, or (3) for both of these reasons.

Most of the cases when they apply to this clinic are clearly classifiable under the list of thyroid diseases—Graves' disease, colloid goiter, adenomatous goiter, thyroiditis, and so forth. But there is a large fraction of cases that cannot be so classified, at any rate, before excision of the thyroid gland. In this class treatment depends entirely on whether the patient can be diagnosed as suffering from *toxicity* referable to the thyroid gland or not. In these cases no treatment is necessary, and no diagnosis of thyroid disease can be made if the patient is not toxic. And again, one is not primarily concerned in these possibly toxic cases as to whether or not they have "toxic adenoma" or whether or not they have "Graves' disease"—the question to determine is whether or not the patient's disability can be referred to the thyroid gland.

The difficulties of diagnosis of toxicity referable to the thyroid are seldom stressed, but the recognition of thyroid toxicity is clearly of as much concern to diagnosticians not directly associated with thyroid clinics as the more discussed problems of therapeutic procedures or the puzzling possibilities of underlying causes of toxicity. Furthermore, correct diagnosis of toxicity within and without a thyroid clinic is a clear factor in the success of the clinic.

It seems evident to us that surgical treatment of thyroid disorders has been worked out, in large clinics, to a highly

satisfactory degree. Surgical death-rate is very low, and this in spite of many cases who are in extreme condition from prolonged toxicity, and others with complicating disorders. It seems doubtful if a much lower surgical death-rate is possible with patients as they are, whatever added ingenuity in preparation and treatment of patients may be exercised. On the other hand, it is clear that many, not all, of the worst operative risks have been through a period when a diagnosis of toxicity could have been made and when they were still excellent surgical risks. And a large part of the deaths are furnished by this group.

It seems evident that early diagnosis and prompt surgical treatment of thyroid toxicity would result in an even smaller death-rate if present standards of surgical methods were maintained. One cannot, of course, avoid the thyrotoxic case who has coincident serious disease which by itself increases their risk.

The Diagnosis of Thyroid Toxicity is Not On a Satisfactory Basis.—The only indirect laboratory test which appears to be at present of much clinical value is basal metabolism estimation, but one must suspect a diagnosis before this somewhat elaborate test is used, and it is only relatively valuable; the final diagnosis has to be made on clinical judgment based on history and physical examination.

It is not possible to lay down rules which if followed will enable anyone to recognize thyroid toxicity accurately or easily. A consideration of the resources which one can call upon to make a diagnosis, and a proper realization of the difficulties seem to us to be the first steps toward overcoming the difficulties.

The routine procedure for diagnosis of thyroid toxicity that we use is simply physical examination and history taking by physician and surgeon. Easily recognizable cases can be promptly classified.

This examination plus a single basal metabolism estimation clearly distinguishes the majority of those that are toxic.

But there remains a "doubtful" group to be divided into:

(*a*) Those that are clearly toxic; (*b*) those that are clearly not toxic; (*c*) not yet diagnosable. These patients are observed for as long as is valuable in hospital. Daily basal metabolism estimations are taken. Repeated physical examination and very thorough history taking is done by surgeons and physicians interested.

Basal Metabolism.—1. It is to be remembered that in general diagnosis basal metabolism estimations are only made after a diagnosis of thyroid toxicity is suspected.

2. To be of value the test must be made in a carefully conducted laboratory. The errors due to faulty technic are well known.

3. Individuals vary greatly in their reaction to the test. Repeated readings on succeeding days show astonishing variations—usually from a higher to a lower reading, sometimes in the other direction. This is true of toxic and non-toxic individuals.

4. Iodin in some form has been taken by nearly all patients before they come to our clinic. This serves further to obscure toxicity and increases difficulties of diagnosis. It seems proved that iodin tends to lower basal metabolism in some toxic cases to some degree.

5. A small but important group of suspected thyrotoxic patients[1] have some complicating disorder which in itself disturbs basal metabolism.

To illustrate the dangers of trusting basal metabolism readings alone in diagnosis: A patient admitted directly to the hospital, and not yet examined clinically, is said to have a basal metabolism reading of plus 35. One has no idea as to whether that patient is toxic. If on examination the patient shows a clear picture of thyroid toxicity, one is satisfied with the basal metabolism estimation and the diagnosis and proceeds accordingly. If one is not sure of the clinical signs, the test is repeated. Individual instances of variation of basal metabolism from clear clinical diagnosis and treatment can be

[1] See article on "Heart Failure (Congestive) Associated with Thyroid Toxicity" in this issue.

quoted ad infinitum. On the other hand, in *statistical reports on groups* basal metabolism estimations have shown clearly that (1) in the toxic states it averages high; (2) in non-toxic states it averages low; (3) in a group of patients clinically changing with surgical treatment from (1) to (2) it first averages high and then averages low. One would not be satisfied to attempt clinical diagnosis of thyroid toxicity without the aid of basal metabolism; nor would any diagnostician dare to depend on this alone.

Discussion of Signs and Symptoms in Thyroid Toxicity from History and Physical Examination.—The age of thyrotoxic patients has ranged in our clinic from three and one-half years to well over seventy years.

Almost 90 per cent. of thyrotoxic cases are females. The 10 per cent. of males, however, disposes of this as a valuable diagnostic aid.

Goiter is not evidence of toxicity. It merely arouses the suspicion of toxicity, and in regions where goiter is endemic it scarcely does that. To be sure, most toxic patients have a goiter, but in individual toxic cases one finds no goiter, or a goiter which may be of practically any size, shape, or consistency.

Eye Signs.—Briefly, *exophthalmos* and *staring* are the two valuable signs clinically. Marked exophthalmos is so striking that a case who shows it can be accepted gratefully as diagnosed already. When definitely present it is almost certain (in cases suitable for consideration) to mean toxicity past or present. Everyone knows, however, that more or less prominent eyes are found in normal people. And occasionally prominent eyes are a cause of error in diagnosis. The observation by a patient that the eyes are becoming more prominent is of possible value when the sign is only by itself suggestively present. There is a large group of toxic cases that do not show enough exophthalmos to warrant a diagnosis made on this point alone. In some cases it becomes a prominent symptom early in the disease, in others late. Many clearly toxic cases do not have it at all.

The most interesting eye sign of value in doubtful cases

is sometimes called *staring*. It is not, as the word suggests, a fixed gaze, but an uncalled for temporary widening of the palpebral fissure noted in the course of an ordinary conversation with a thyrotoxic patient under examination—similar to widening which occurs when a normal individual is particularly startled. This sign speaks very strongly in favor of thyroid toxicity in the doubtful case, though it can be occasionally noted in people with uncomplicated neurasthenia. Many toxic cases do not have it clearly enough to enable the examiner to be certain of it. Others do not have it at all.

Clearly, these signs are of a relative value which increases absolutely only with the personal experience and judgment of the examiner.

Pigmentation, as described by Jellinek, a brownish staining confined to the eyelids, is an occasional sign quite inconstant in thyroid patients and sometimes present in individuals without thyroid toxicity. It is haphazard in its time of appearance and in its selection of individuals. A larger proportion of the prolonged severe cases, particularly those with congestive heart failure, show a generally distributed brownish staining of the skin. This is a striking sign when present, but, unfortunately, is not to be distinguished clinically so far as is known from pigmentation sometimes associated with other chronic conditions. And if there is some inherent difference, the differential diagnosis has not yet been developed to a useful degree.

Myasthenia—an actual, often sudden, apparently muscular failure under ordinary strain, for example, the knees giving way and the patient falling when climbing stairs—has often been stressed as a symptom of thyroid toxicity. It is a fairly common story. And yet we can recall no cases where it has been of great value in diagnosis in a doubtful case.

Gastro-intestinal Complaints.—Sudden diarrhea or attacks of vomiting lasting for several days or several weeks, with critical relief, are apparently not commonly present in truly doubtful cases, but an occasional severely sick but doubtfully toxic individual has shown them as a prominent and early symptom. The possibility of a hyperthyroidism should be carried in the

back of one's mind in the presence of such an occurrence not otherwise clearly explained.

Loss of weight, weight fluctuations, and *chronic emaciation* are very valuable signs. Unaccountable loss of weight serves to suggest a thyroid toxicity by itself, and it is commonly present. Only rare cases are able to hold their weight constantly and very rarely can a thyrotoxic case gain. But fluctuations in weight with fluctuations in toxicity are not uncommon. A fairly large fraction of prolonged cases are emaciated—weigh less than 110 pounds and are unable to recall a time when they weighed appreciably more. Chronic emaciation is, perhaps, as valuable in diagnosis as a distinct rapid loss of weight. One of these three points is nearly, but not always, to be made from the history of a thyrotoxic patient. One hesitates to diagnose toxicity in their absence unless other signs are clear. An occasional severely toxic case is able to gain to above previous and standard weight.

Increased fatiguability is a very constant finding, but an occasional case shows nothing to suggest it. A definite number of the severely toxic cases have carried on with their full duties to an astonishing degree until they have come to us, and have come because of the unsolicited advice of friends or physicians, who noted the patient's condition, though the patient was unconscious of it.

Undue breathlessness is a vague term which cannot be considered except by a too exhaustive discussion.

Vasomotor disturbances, such as sweating, flushing, sensation of heat, dislike of warm weather and places, are common, particularly discomfort in warm places. This last complaint is strongly suggestive. But again, it is rarely more than a valuable hint in diagnosis of doubtful cases, and its complete absence does not disturb a diagnosis. Nor are the other signs very helpful in our experience.

Emotional Disturbances.—Long association with thyrotoxic cases familiarizes one with tiny habits or emotional reactions, such as easily elicited tears with equally prompt return to mental equilibrium, or occasionally an overstimulated rapidity

of thought and conversation. On the other hand, the neurasthenic's conversation often helps to suggest the true diagnosis by an overemphasis of simple complaints. Phobias, sleeplessness, headache, sensation of pressure in the back of the head or forehead are common complaints of neurasthenics and are not usually mentioned by purely thyrotoxic individuals. But there is no certainty about the emotional states in thyroid toxicity. Many of the most toxic cases show apathy and mental and physical hebetude distinctly opposed to the supposedly characteristic picture of mental and physical restlessness and overactivation. And in doubtful cases mental and physical "nervousness" appear more properly as the cause of a patient's coming for advice than as a helpful suggestion in differentiating thyroid toxicity from neurasthenic or fatigued states. Both conditions are often found in the same individual in a thyroid clinic.

Tremor.—This "cardinal sign" of thyroidism is so commonly present in neurasthenic individuals, or patients sick from any cause, and also in entirely normal individuals, that in our experience (with increasing observation) it has grown less and less impressive. Its absence or presence is a matter of little interest. It is, however, rarely absent in the "overactivated" thyrotoxic individuals.

Thyroid Heart.[1]—Briefly, tachycardia, rapid heart-rate, is almost without exception present. A regular rate below 80 at ordinary examination almost serves to rule out an underlying thyroid toxicity. There are, however, definite exceptions to this rule.

Auricular fibrillation, transient or paroxysmal, or as an established condition is present in about 20 per cent. of all toxic cases and its percentage incidence increases from no per cent. of those under twenty years of age to 85 per cent. of the cases over seventy, varying somewhat within each age decade with degree of toxicity. Its presence in a patient not clearly deserving it from an underlying heart disease should suggest the possibility of thyroid toxicity.

[1] See article entitled "The Heart in Toxic Thyroid States" in this issue.

There are no other signs of value in the diagnosis of the thyroid heart *per se*. Enlargement is not present in uncomplicated cases except rarely. Forceful beating, shown by an easily felt thrust, sometimes even causing movement of the thoracic cage, and overpulsating arteries, though common, are not always demonstrably present, and are, of course, not peculiar to thyroidism.

The presence of small amounts of *glucose*, four-tenths or two-tenths of 1 per cent., *in routine urinalysis* is very common in hyperthyroidism, but it is not constant. It is common enough in other conditions to exclude it as of value in diagnosis of doubtful cases.

Menstrual disturbances in the direction of amenorrhea are common, but not invariably present.

Common Errors in Diagnosis of Thyroid Toxicity.—Close to 30 per cent. of all cases referred to the thyroid clinic fail to show any disease referable to the thyroid gland.

By far the largest portion of this group have no disease whatsoever, but are disturbed in large part by the symptoms described above as increased fatiguability, tremor, rapid heart, and by emotional and vasomotor disturbances. These cases of "neurocirculatory asthenia," "effort syndrome," "neurasthenia," whatever they may be called, offer the greatest problem in differential diagnosis.

It is well to remember that the medical profession as a whole became conscious of this large group of "neurocirculatory asthenic" individuals during the World War, and as a result of war-time examinations, where diagnoses were subject to checking by many individuals. And that men of the highest professional standing when they first saw these cases uncovered by the war in large numbers inevitably believed first that they were dealing with cases of heart disease, and then that they were dealing with hyperthyroidism. It is impossible to avoid this group of cases in clinical work. It is unfortunate that they are difficult to describe and to teach to students, and that they are particularly resistant to treatment. And that in the mass of detail that one must remember to avoid gross errors in or-

dinary diagnosis of serious conditions, one is inclined to leave these troublesome cases in the background of medical discussion and study. One can at least remember that they exist and are frankly confusing by the difficulties of diagnosis that they present.

We have found that it gives a misleading impression of finality to attempt a list of symptoms commonly shown by clearly thyrotoxic patients, in distinction to those shown by typical neurocirculatory asthenics, just as it does to speak of "normal" and "elevated" basal metabolism estimations. Actually, only long clinical experience with these problems enables one to value laboratory tests and clinical signs and symptoms to any satisfactory degree. It is perhaps unfortunate, but true, that the most important thing in the diagnosis of thyroid toxicity in doubtful cases before surgical treatment is "who says so."

The above is the only large group of individuals that are at all commonly *wrongly* suspected of thyroid toxicity. In the course of observation of a long series of cases referred for thyroidism, one finds a few individuals suffering from one of a variety of diseases. An occasional wrongly suspected thyrotoxic case proves to have diabetes. Of course, an occasional active tuberculosis or chronic nephritis is wrongly suspected of thyroid toxicity. The overwhelming majority, however, of all wrongly suspected toxic thyroids prove to have nothing of significance except a neurasthenic state.

In the course of study of the truly thyrotoxic patients one finds a large number previously suspected and treated for one or more of a great variety of conditions.

By far the largest group of undiscovered thyrotoxic cases has been previously treated for "heart disease." The most dramatic cases in this group are those described in my article in this issue dealing with true congestive heart failure caused by hyperthyroidism.

A most unfortunate confusion is that of wrongly diagnosing a thyrotoxic patient as a diabetic.

Extraordinary case histories could be cited where abdominal surgery has been employed elsewhere because of a persistent

vomiting from a severe atypical thyroid toxicity without obvious physical signs suggesting thyroid disease.

There is another group of thyrotoxic cases where the apparent diagnosis was pulmonary tuberculosis, and the patients have been treated accordingly for long periods.

Amenorrhea and vomiting as the prominent symptoms in a rare thyrotoxic young woman with obscure signs of toxicity clearly may be misleading.

In a thyroid clinic, with patients in hospital, under examination by a number of physicians and surgeons experienced in thyroid disorders the majority of the group of doubtfully toxic cases can safely be separated into: (1) clearly toxic, (2) clearly not toxic. There remains a small group of individuals—"not yet diagnosable"—who must be followed sometimes for long periods before the diagnosis can be proved. In our experience most of this group have later proved to be toxic.

One can lay down no rules for the easy and early recognition of thyroid toxicity, but experience with many individuals shows clearly that:

Thyroid toxicity is of haphazard onset. It may come to any patient at any time. Its symptoms are variable; they follow no set course. Many cases never become clear cut. It may simulate a variety of disorders, particularly heart disease, tuberculosis, diabetes. It may simulate a nausea and vomiting of pregnancy, or an acute surgical abdominal condition. *It is rare. Many of its common symptoms are common to the commonest of all complaints, neurasthenic states.*

It seems to us that an alert diagnostician must suspect thyroid toxicity wrongly many times. If he carefully confirms his diagnosis the occasional correctly suspected and proved case will be a source of much satisfaction to patient and diagnostician. There are few causes of chronic disability that yield so clearly to treatment.

SIMPLE SEROUS CYSTS OF THE KIDNEY

HOWARD M. CLUTE

SIMPLE serous cysts of the kidney are surgical curiosities. Harpster, Brown, and Delcher were able to discover but 95 cases

Fig. 538.—Case I. Schematic drawing, showing serous cyst arising from lower pole of kidney. Cyst removed. Kidney saved. Recovery.

recorded in the literature after a careful search. Three recent cases in this clinic are worthy of note.

The first patient, a thin, poorly nourished woman, came to the clinic for treatment for "falling of the womb" and "a tumor in her side." On examination it was found that she had a second degree prolapse of the uterus and a lacerated perineum. In the right lower quadrant of the abdomen a mass the size of a grape-fruit could be felt. This mass seemed to arise in the

Fig. 539.—Case I. Pyelogram in prone position, showing outline with arrow of cyst at lower pole of kidney.

region of the kidney and was quite freely movable. It was fluctuant It was not tender and motion caused no pain. Cystoscopy was done and pyelograms were made. On the right side the kidney was rotated and low. Some obstruction was present, as shown by the dilated renal pelvis and blunted calices. Below the kidney a vague, round shadow was seen, to which we at-

tached little significance at this time. The prolapse being the chief cause of the patient's difficulties, a vaginal repair and suprapubic suspension of the uterus were done. At this time a large cyst was found attached to the lower pole of the right kidney. On a later date a second operation was performed for

Fig. 540.—Case I. Pyelogram showing descent and rotation of kidney pelvis from the weight of the serous cyst. This picture was taken with the patient standing.

the removal of this cyst. An incision was made in the right loin and the kidney readily delivered. Arising from the lower pole with a wide area of attachment was a thin-walled cyst filled with clear serous fluid. There was no connection between the cyst and the pelvis of the kidney. The cyst was readily peeled off from the surface of the kidney, although fairly free

Fig. 541.—Case II. Drawing of kidney with two serous cysts. Cysts removed. Kidney saved. Recovery.

bleeding occurred from the bared renal tissue. This hemorrhage was readily controlled by the actual cautery and by

sutures passed through the kidney substance. The patient made an excellent recovery and is now apparently quite well.

The second and more recent case occurred in a stout man sixty-three years of age, who came to the clinic because of a "sense of fulness in his abdomen, pressure on his stomach, and a swelling in the abdomen." Examination showed a tumor in

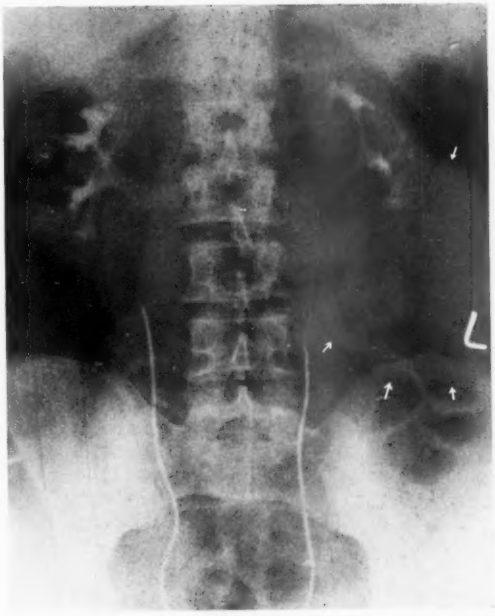

Fig. 542.—Case II. Pyelogram showing large spherical cyst arising from lower pole of kidney and not communicating with the pelvis of the kidney.

the left upper quadrant of the abdomen the size of a baby's head. This tumor was very slightly movable, did not move with respiration, and it was slightly tender. Cystoscopy revealed a normal bladder save for early prostatic hypertrophy. Pyelograms revealed a normal pelvis on each side. On the left side was seen a large circular swelling apparently overlying the lower

pole of the kidney. It did not communicate with the renal pelvis. At operation a large simple serous cyst of the lower pole of the left kidney was found. It was readily removed by cutting away the sac of the cyst. At first the layer of cyst attached to the kidney was left in place in an attempt to obviate the hemorrhage that followed its removal in the former case.

Fig. 543.—Case III. Pyelogram of large cyst of left kidney. Some ureteral obstruction and extrarenal dilatation of the pelvis. No infection. No symptoms. No operation.

This procedure was deemed inadvisable on further consideration, and the entire serous coat was removed and the kidney sutured. On the posterior surface of the kidney was a similar though much smaller cyst the size of an egg, which was treated in the same manner. The patient made an excellent recovery from the operation.

The third case occurred in a woman sixty-four years of age,

who complained of "diabetes and a swelling in the abdomen." On examination this patient showed a tumor in the region of the left kidney that appeared about 8 inches in diameter. It was round in outline, freely movable, and fluctuant. With respiration it moved but slightly. It was not tender and caused no distress. There were no urinary difficulties and repeated urine examinations were entirely negative for pus. Pyelograms on this patient showed a rounded tumor arising from the lower pole of the left kidney and displacing the ureter toward the spine. The renal pelvis was dilated, apparently from pressure upon or distortion of the ureter. There was no evidence of renal infection.

Operation in this case was not advised because of the patient's diabetes, her age, and because of the fact that the cyst was causing no discomfort and offered no immediate possibilities of serious complications.

The origin of simple serous cysts of the kidney is not known. Older writers have thought that the cysts arose from a developmental defect resulting in incomplete fusion between the malpighian bodies and the connecting tubules. Most writers in recent years have favored the retention theory, and have reasoned that the cyst arose because of some inflammatory or congenital obstruction of certain of the kidney tubules.

The pathologic report on the specimens removed in the 2 operated cases as given by Dr. Lawrence Smith is as follows:

Case I.—Microscopic description: Sections microscopically throughout the wall of the cyst present evidence of kidney tissue undergoing pressure atrophy. Most of the tubules have entirely disappeared in this area. A few of the glomeruli are visible. The vessels appear hyalinized. A few foci of necrosis with hemorrhage and calcification are noted. There is a suggestion of beginning arteriosclerosis in the rest of the kidney. No evidence of tumor is found. Diagnosis: Simple cyst of the kidney.

Case II.—Microscopic description: Sections through the wall of the cyst, microscopically, show a dense fibrosis in which

considerable hyalinization has taken place. Some further degeneration of the tissue has occurred, with a resultant secondary calcification. Such kidney as persists presents evidence of pressure atrophy in the areas near the cyst. There is a definite sclerosis of the vessel in these areas, with secondary degeneration of the glomeruli and tubules supplied by them. No evidence of malignancy found. Diagnosis: Simple cyst of the kidney.

Serous cysts of the kidney give rise to no symptoms until they reach sufficient size to cause pressure upon neighboring organs. They may, by dragging on the kidney, so distort the pelvis and ureter that ureteral obstruction and pelvic dilatation occur, as was seen in the pyelograms of the first and third cases. The presence of a slowly increasing abdominal tumor may, however, cause the patient no little alarm. For this reason, if for no other, their proper diagnosis and treatment is imperative.

The positive diagnosis of this condition is not always possible. The exclusion of hydronephrosis or pyonephrosis can be made by finding a normal pelvis and a normal renal outline in the pyelogram. The absence of urinary symptoms is suggestive. The appearance of a symmetric swelling arising from one pole of the kidney separate from or overlying a normal pelvis is the typical finding in the pyelogram. The ureter may or may not be displaced or obstructed by the cyst, with resulting dilatation of the renal pelvis.

The treatment of simple serous cysts of the kidney is removal of the cyst. In the past many kidneys have been sacrificed in this condition which could now be saved. The cyst wall can be peeled from the surface of the kidney very readily, just as the lining membrane of an ovarian or thyroid cyst can be removed from the ovary or thyroid. The hemorrhage, while moderately brisk in each of these cases, was readily controlled by ties and sutures. A small drain may be needed to take care of the dead space left after removal of the cyst.

Conclusions.—1. Simple serous cysts of the kidney are rare. They cause no symptoms until they reach sufficient size to produce pressure on neighboring organs.

2. The diagnosis is made by finding a freely movable, benign, fluctuant tumor in the kidney region, often visible by *x*-ray and not connected with the kidney pelvis in the pyelogram.

3. Serous cysts of the kidney can be shelled out from the kidney in many cases and the kidney preserved. Nephrectomy may occasionally be necessary.

NON-CALCULOUS URETERAL OBSTRUCTION

HOWARD M. CLUTE

OBSTRUCTION of the upper urinary tract by kinks or distortions of the ureter undoubtedly occurs with sufficient frequency to render the diagnosis of this condition a matter of

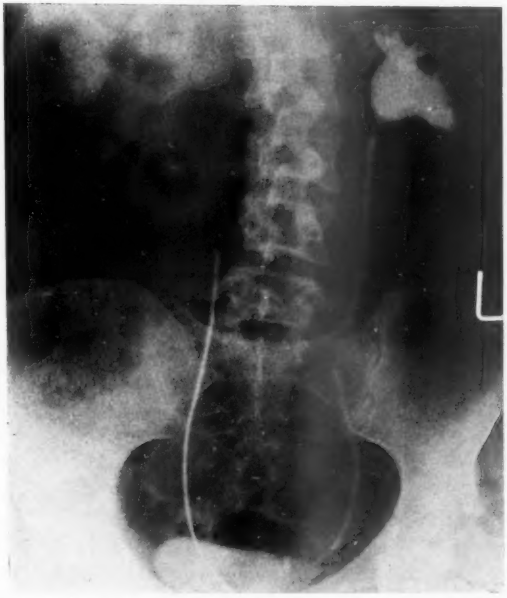

Fig. 544.—Obstruction at ureteropelvic junction. Improved by ureteral dilatation.

definite importance to medical men. The great difficulty today in dealing with possible cases of this kind lies in the correlation of symptoms and physical examination, and in the decision as

to the proper treatment to employ. The decision in any pyelogram as to what really constitutes a ureteral stricture or kink is often dependent on the individual observer's judgment. Moreover, to determine that a given narrowing or variation in the course of a ureter is responsible for the patient's symptoms

Fig. 545.—Kink at ureteropelvic junction with dilatation of renal pelvis above. Symptoms resembled an acute attack of appendicitis. Operation—pyelotomy and plastic closure of pelvis with suspension of kidney. Cure.

requires much careful consideration, and is by no means an easy problem. The operative attempt at removal of supposed kinks of the ureter should not be lightly undertaken.

The clinical picture presented by ureteral obstruction is dependent on the degree of obstruction and the rapidity of its development. The leading symptom in all types of obstruction

to the ureter is pain. The location and the degree of this pain is extremely variable. Most often it occurs on the affected side, in the upper quadrant of the abdomen. It may be most severe over the kidney and radiate either to the front of the abdomen or down the course of the ureter into the groin. The pain usually recurs in attacks of varying intensity. Rarely the patient states

Fig. 546.—Ureteropelvic kink from aberrant vessels and fibrous bands. Operation refused at this time. Dilatation of no benefit.

that he is never free from a sense of distress or pressure in the abdomen. Tenderness over the kidney is unusual. Tenderness over the ureter is not infrequent and undoubtedly accounts for many abdominal operations done in these cases before the correct diagnosis is made.

Fever has been absent in all cases we have seen when this condition was suspected. Nausea and vomiting occur if the

obstruction is so sudden and so complete that the resulting pain is of great severity. When this type of obstruction occurs the picture resembles an acute intra-abdominal surgical emergency. Pain again is the leading symptom. It may be located over the kidney or the gall-bladder or it may extend down the ureter.

Fig. 547.—Pyelogram, same case as shown in Fig. 541, ten months later. Some slight increase in pelvic dilatation. Deformity still present. Operation. Cure.

Well-marked muscle spasm and tenderness are likewise found. The patient frequently has nausea, vomiting, and more or less collapse.

Examination of the urine in cases of ureteral kink with obstruction very often is entirely negative. The occasional case shows a few red blood-cells or pus. In the bulk of our cases

nothing remarkable was present in the urine. Frequent and painful micturition is often present. No relation is noted between the presence of the pain and urination in very many cases.

The group of patients suffering from ureteral obstruction, acute in onset and more or less complete in degree, present a most interesting picture. Of this type we have seen two ex-

Fig. 548.—Ureteral kink with pelvic dilatation above. At operation a thick fibrous band was found adherent to ureter.

amples to which brief reference may be made here. In each of these cases the onset was marked by severe pain over the right side of the abdomen. This was followed by nausea and vomiting. There was no fever. Examination showed general abdominal tenderness in the right lower quadrant in each instance. In one case there was slight tenderness in the right costovertebral angle. The urine in both cases was entirely negative.

In the first case the abdomen was opened by a right rectus incision and a normal appendix was found. The right kidney pelvis was found markedly enlarged and tensely dilated. Its contents could not be expressed nor could any stone be felt. The posterior parietal peritoneum was opened and the ureter and pelvis closely examined. A definite, thick, fibrous band, obstructing and elevating the ureter, was found and cut. The

Fig. 549.—Ureteral kink due to bands distorting ureter and giving attacks of intermittent obstruction. Operation. Recovery.

contents of the pelvis now quickly escaped through the ureter. Nothing more was done. The abdomen was closed in layers and the patient made an excellent recovery. He has had no further trouble after four years.

In the second case, after considerable deliberation, two careful urine examinations, and a negative *x*-ray of the urinary tract, operation was undertaken for appendicitis. On opening

the abdomen a normal appendix was found and removed. Further examination showed the pelvis of the right kidney to be markedly dilated and tense. Its contents could not be expressed. No stone or obstruction could be felt. Nothing further was done at this time because the patient's condition was unsatisfactory. A week later cystoscopy was done and pyelograms were made. These revealed a well-marked dilatation of the right kidney pelvis, with an apparent kink at the

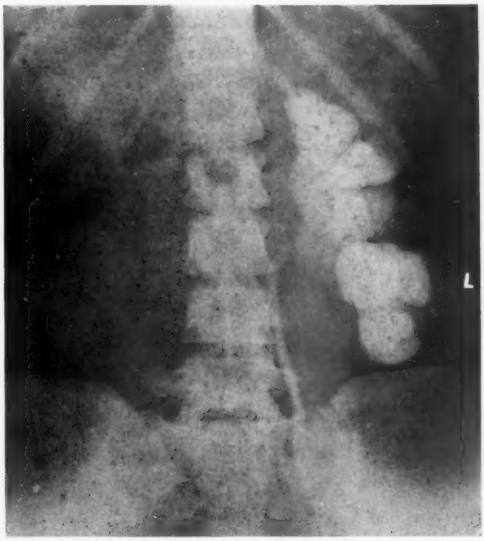

Fig. 550.—Bilateral hydronephrosis from ureteropelvic obstruction. Plastic operation on the right. Improvement.

ureteropelvic junction. Fourteen days after the first operation the right kidney was explored. No evidence of kinking or stricture of the ureter could be found. No obstructing bands were present. The pelvis was opened and it was found that sounds passed to the bladder readily. The pelvis was sutured and the kidney carefully suspended. The patient made an uneventful recovery and has been free from all pain for the eight months that have passed since operation.

These 2 cases are unusual results of ureteral kinks, with sudden and fairly complete blocking of the ureter. The cause of the obstruction could not be demonstrated in the second case.

The treatment of ureteral kinks must vary with the type of kink, the degree of obstruction, and the symptoms that are produced. Patients complaining of vague abdominal pain, with apparent kinks of the ureter, but with no dilatation of the renal pelvis or ureter above the kink, should be given a thorough trial

Fig. 551.—Bilateral hydronephrosis from ureteropelvic obstruction. Plastic operation on the right. Improvement.

with conservative methods. Cystoscopy should be done at intervals and pyelograms made in order to ascertain with certainty that the ureter is strictured or kinked constantly at a given point. Failure of a kink to persist at different examinations makes its presence unlikely. If the strictured point is found at the same level on two or more examinations, it may be considered as a true lesion of the ureter. Kinks and strictures in the course of the ureter can be helped by the passage of a

ureteral catheter. This relieves the patient of all pain in a few cases. At times it is necessary to pass two catheters at once into the ureter at repeated intervals and thus dilate the stricture. Bougies or Garceau catheters may be used for the same purpose. In some cases the ureteral catheter has been left in the ureter for several hours or even two or three days. This apparently straightens out the ureter and relieves the symptoms.

Fig. 552.—Kink in upper ureter. Symptoms cured by operative removal of obstructing bands.

Why this should have any effect on the kinked ureter is impossible to explain.

Surgical operations upon apparent kinks of the ureter with ill-defined symptoms and no dilatation of the pelvis above should be undertaken with great hesitancy, as the surgical findings in such cases usually are far from satisfactory in explaining the condition. Conservative methods should always be

given a thorough trial in these cases. Not infrequently an intra-abdominal lesion, such as salpingitis, will be found which is definitely related to the strictured ureter. Such a lesion must have precedence in the scheme of treatment.

Kinks of the ureter with blunting of the calices of the kidney or dilatation of the pelvis or ureter above the kink more fre-

Fig. 553.—Infantile kidney on the left with ureteropelvic constriction. Apparently improved by dilatation.

quently require operative measures for relief. Conservative measures may be tried in such cases, but are less likely to produce lasting results. When cystoscopic instrumentation fails to give relief from the pain an operation should be done upon the kidney and ureter. In a few cases the kidney has been exposed by an anterior incision. This has given an excellent opportunity to examine the ureter in its course and at its junc-

tion with the renal pelvis. Any obstructing bands that can be found are severed. Usually, however, the kidney is approached through the loin. It is important to follow the ureter well down in its course in order to remove any obstruction. The ureteropelvic junction must be carefully examined as to the angle between the ureter and pelvis. In one case in which the ureter entered the pelvis near its upper end a plastic operation was

Fig. 554.—Apparent kink of upper ureter with pelvic dilatation. See next cut showing stricture in lower ureter which is the real cause of the obstruction and dilatation of the ureter.

performed which converted the pelvis into the normal funnel shape. One need not hesitate to do a pyelotomy and dilate the ureter with sounds if no external bands can be found causing obstruction. After removal of the obstruction the kidney is suspended as high as possible. Care must be taken not to rotate the kidney during this procedure.

Conclusions.—1. Ureteral kinks, usually requiring conserva-

tive treatment, should be carefully differentiated from surgical emergencies.

2. Symptoms and signs of ureteral kinks depend upon rapidity of development and degree of resulting obstruction. Pain is the most constant symptom. Urinary changes are unusual.

Fig. 555.—Stricture obstructing lower ureter possibly secondary to salpingitis. Kink seen above is not obstructing. Symptoms of renal pain disappeared after salpingectomy. These plates show the need of filling the whole ureter in making pyelograms.

3. Two cases treated surgically for supposed acute abdominal conditions are discussed. In both cases acute ureteral obstruction was the cause of the condition.

4. The treatment of ureteral kinks should be conservative unless there is dilatation of the pelvis or ureter above the kink, or unless instrumentation fails to relieve the pain.

COMPLICATIONS SUBSEQUENT TO THYROIDECTOMY

HOWARD M. CLUTE

ALL surgical procedures carry with them a certain risk which fundamentally is due to either the effect of the disease undergoing treatment or to the actual surgical maneuvers carried out. This fact is particularly obvious in thyroid surgery. Only by special attention to both the severity of the thyroid lesion and the surgical technic employed to cure it can the present good results that are obtained be made certain.

No other group of patients require more particular attention by men interested in the different branches of medicine than do those suffering from thyroid disease. The surgeon must work in close association with the anesthetist, the internist, and the laboratory worker. Thyroid disease cannot to the best advantage be diagnosed, treated, and followed up save in a clinic accustomed to this type of case and organized to deal with it.

No small part in the eventual good recovery of the thyroid case after operation lies in the postoperative care which it receives. The difficulties and complications that may arise after operation are in many respects similar to those that may follow any surgical procedure. Hemorrhage or infection in a wound in the neck assumes a vastly different significance than in most other parts of the body because of the anatomic relations of the area involved. In addition, there is, in thyrotoxicosis, the decidedly unusual factor of the postoperative reaction of the patient to any operative procedure.

Frank infection of the neck after thyroidectomy is unusual. The collection of clear or blood-stained serum under the skin-flap is not infrequent. This serum is due to the relatively large area of superficial tissue dissected when the skin-flap is

raised, to the presence of many bits of catgut ties immediately under the skin, and to the presence of a small amount of blood-clot. Often small amounts of serum will be entirely absorbed if left alone. Usually it is wiser to open the scar with a probe on the fourth or fifth day and release the serum from the wound. This procedure may have to be repeated several times in a single case. Drainage, if it can be avoided, and it usually can, is not advisable because of the danger of subsequent infection. At times the postoperative fever, which is frequently present on the first and second day after operation, will persist to the third or fourth day. This usually means infection in the wound and always demands wound inspection. If the skin-flap is red, swollen, and unduly tender, it should be opened at once in the scar. Frank pus will sometimes be present. In this case rubber-dam drainage should be inserted at once. It is important to discover this type of infection early in its course and institute proper treatment. At times a cellulitis in the wound may occur. The upper portion of the wound is brawny and indurated, with moderate tenderness. This induration may extend laterally or upward under the jaw. Rarely if ever does it extend down over the chest. The lymph-nodes along the jugular veins and in the submaxillary triangles may become indurated and tender. Considerable fever is usually present. Such a wound does well with free drainage and local heat. This may be applied in the form of frequent hot boric acid compresses or hot flaxseed poultices.

Of all infection after thyroid operations, that which extends downward into the thorax (mediastinitis) is the most serious. Mediastinitis is a rare complication of thyroid surgery, but when it occurs it is very apt to be fatal. Mediastinitis most frequently follows the removal of colloid adenomatous goiters which have extended downward into the superior mediastinum. It develops slowly and with no characteristic symptoms. A persistent low fever is present. The wound shows evidence of a low-grade infection with slight but constant purulent discharge. Cough and difficulty in breathing often occur, resisting all methods generally useful for their control. Marked

interference with swallowing follows as the infection approaches the course of the esophagus. Severe choking fits are not infrequent and pain is so marked on swallowing that nutrition suffers. The patient acquires a markedly cachectic appearance, grows rapidly weaker and more apathetic, and dies several weeks after the onset of the infection.

Treatment for mediastinitis is decidedly unsatisfactory. Every attempt should be made to prevent its occurrence by gentle manipulation inside the thorax and adequate drainage of all substernal pockets at the time of operation. If this complication occurs, no local measures will cure it.

Hemorrhage after operations on the thyroid gland is of serious importance because of the large amount of blood that may be lost in a short time, and because of the dangerous effects of rapidly increasing pressure on the trachea. Serious bleeding comes, in general, from either the superior thyroid pole or from the anterior jugular veins on the prethyroid muscles. Postoperative bleeding from the superior thyroid artery is a serious emergency and demands immediate attention. It most frequently occurs soon after operation from the slipping of a ligature about the superior pole. A large hematoma forms quickly under the prethyroid muscles, and symptoms of tracheal pressure and obstruction follow. The neck must be opened widely, the muscles cut, and the superior pole isolated, caught, and tied. Hemorrhage from the superior pole occasionally follows infection of the wound and sloughing about the artery. Here, again, the neck must be widely opened and the bleeding vessels carefully exposed. In the presence of much slough the superior thyroid artery must be followed up into clean tissue to its origin on the external carotid artery and tied in continuity. Packing and pressure are of little avail and, if employed, result only in a recurrence of the hemorrhage in many of the cases, the vessel finally being adequately exposed and ligated at its origin in a patient ill prepared to withstand any surgical procedure. Only radical measures are safe.

Hemorrhage from the anterior jugular vein is much less serious and is easier to control. It shows itself by slow leakage

from the wound and the formation of a hematoma under the skin-flap. The wound should be widely opened, the hematoma carefully removed, and the bleeding vessel found and tied. When the neck is opened and the clot removed, it may be that no single bleeding point will be discovered. The wound should be closed with a drain.

In certain large colloid or adenomatous goiters a large amount of blood will be lost during operation. This is at times unavoidable. During all operations in the clinic the anesthetist keeps a record of the pulse, blood-pressure, and respiration of the patient. When much blood is lost the pulse-rate rises rapidly and the blood-pressure drops suddenly. This record of the anesthetist is invaluable in helping to decide when a patient has lost so much blood that a transfusion is indicated. One must be on the safe side and give an immediate blood transfusion when any doubt exists. Such a transfusion not only has a great immediate benefit but also is of value to the patient during convalescence.

When the thyroid is retrotracheal or substernal, or when the trachea is bared in its removal, more or less tracheal irritation will occur postoperatively. This is marked by pain under the sternum in the course of the trachea, cough, and some dyspnea. The cough is often difficult to control and may be a great annoyance to the patient. The sputum is often blood tinged. Rarely the respiratory rate may be elevated and bronchitis or bronchopenumona, with slight fever, may be present. In cases with marked tracheitis or bronchitis the wound should be carefully inspected for any collection of fluid that may be causing tracheal pressure. Drains must be removed and replaced by smaller ones. A steam tent which permits plenty of fresh air saturated with moisture from fresh steam is valuable in these cases.

Occasionally marked dyspnea and tracheitis occur after the removal of goiters which by their pressure have distorted the shape of the trachea. The rings may be flattened on one or both sides and the lumen correspondingly narrowed. The trauma necessary to the removal of the goiter and the loss of

its support may cause partial obstruction of the trachea from either edema or collapse. These patients usually show well-marked dyspnea and tracheitis soon after operation, but respond well to treatment with the steam tent and the erect posture. Very rarely breathing will become so laborious that the patient is rapidly tired out and cyanosis may appear. It is extremely important that tracheotomy be done as soon as these symptoms are apparent. It is a great temptation to delay such a tracheotomy too long. A patient who is obviously struggling for her air may still maintain quite good color with only an occasional bit of cyanosis. Slight cyanosis with difficult breathing means obstruction of the air-way, and only by great effort does the patient maintain sufficient oxygenation of her blood. Due to tiring efforts and to the insufficient oxygen intake the patient grows rapidly weaker. She later becomes sleepy, and finally loses consciousness, even though her cyanosis be little more than noticeable. Tracheotomy done at this last stage will often be of little value, the patient never regaining consciousness. Tracheotomy done when cyanosis first appears produces a remarkable improvement in every respect. It should be employed rather than delay when one is in doubt.

Injury to the trachea rarely occurs during operation, but when such an accident does occur, unless handled properly it may be extremely serious due to sucking in of blood into the trachea. It may be cut with a knife or pinched in a snap. Such an injury must be immediately sutured and the wound closed with a small drain. Usually no further trouble will result. Should dyspnea or cyanosis follow this accident, a tracheotomy should be done without delay.

The accentuation of all symptoms of hyperthyroidism after operation on toxic goiters is a complication which all thyroid clinics know and fear. This occurrence is generally called a thyroid storm. It is best treated by preventing its occurrence through extreme care in selecting the operative procedure for the individual case that will be well inside the limit of safety.

The onset of a thyroid storm may occur from six to thirty-six hours after operation—most frequently ten to twelve. There

are two general types of thyroid reaction. The first type is one of great activation. All the symptoms of the disease are present in an exaggerated form. The pulse-rate, already high, becomes higher, occasionally rising to 240. A persistent rate of over 200 should be looked upon as a very unfavorable symptom. The nervous symptoms become greatly exaggerated. Tremor of the hands and arms with constant muscular activity is pronounced. The eyes are staring and the facial expression shows marked apprehension. Rest is impossible and the patient tosses constantly about the bed. The skin is hot and moist with perspiration. The temperature is always elevated from 1 to 4 or 5 degrees.

The second type of thyroid storm is marked by great depression and resembles in some respects surgical shock. The pulse-rate is persistently high and the temperature is also elevated. The patient, however, shows no accentuation of her nervous symptoms. Frequently she cannot be roused, but lies quietly in bed with her eyes closed or partly closed. Her skin is not hot and her face is not flushed, but white. She shows no tremor and no marked restlessness.

The treatment of the first type, which is the more common, can be given under two headings—morphin and fluids. Morphin must be given in sufficient doses and with sufficient frequency to keep the patient quiet. Much larger doses are required here than in non-toxic cases, and the drug must be administered until the desired effect is obtained. Occasionally codein has had more value than morphin in controlling the incessant activity. Fluids are also given freely by mouth and by rectum or subcutaneously, or given as a routine to all patients who are expected to show a marked thyroid reaction.

The depressed type of thyroid reaction requires little if any morphin. Subpectoral and intravenous fluids are very valuable.

Conclusions.—1. The complications which may follow thyroid gland operations are those common to all surgical procedures. They acquire especial significance because of the location of the wound in the neck.

2. The most serious result of infection in the thyroid wound is mediastinitis. This complication usually results fatally.

3. Hemorrhage after thyroidectomy requires immediate and radical surgery for its control.

4. Tracheotomy should be done at once when any marked obstruction to breathing occurs after operation.

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TREATMENT OF COMMON DUCT BILIARY FISTULÆ BY ANASTOMOSING THEM INTO THE INTESTINAL CANAL

FRANK H. LAHEY

IN the issue of The Journal of the American Medical Association, March 31, 1923 we published a method of dealing with complete biliary fistulæ by dissection and preservation of the fistulous tract and by anastomosing or implanting the tract into the duodenum.

After having employed this method in two further cases we wish to call attention again to its advantages, and mention its disadvantages, as a resort in cases where the common duct has been largely destroyed, or where it is not feasible either to unite two cut ends of the common or hepatic duct or to connect the end of the hepatic duct with the duodenum.

Where complete loss of substance of an entire section of a common duct occurs, one of the following procedures is possible: an end-to-end anatomosis of the cut ends of the cut, with considerable possibility of stricture occurring at the point of union; a rubber tube may be implanted, so that either end of it rests within the cut ends of the duct and the tube bridges the gap between the two cut ends. In such a procedure the omentum may be wrapped about the tube in an endeavor to establish a bile tract, or the rubber tubing may be left free to become encysted, and thus establish its own tract. Where no lower end of the duct is present or discoverable, the bowel may be brought up to the open end of the hepatic duct, and an attempt made to produce a direct anastomosis between the bowel and its open end; or the end of the hepatic duct may be connected with the duodenum by a segment of rubber tubing. The last, and perhaps the least feasible, procedure is the fashioning of a new duct from the muscular wall of the stomach by

converting a rectangular gastric flap into a tube and anastomosing it to the upper end of the duct.

The operation here suggested, that is, implantation of the fistulous tract into the duodenum or stomach, depends for its success or failure largely upon the same factors governing success or failure in the above-mentioned measures, but in our experience it is technically much easier and safer to perform.

The drawbacks to all of the operations for restoration of the biliary canal are: (1) Cicatricial contraction of the new scar tissue canal, and (2) infection within the biliary passages due to loss of the sphincter of Oddi.

In a very large majority of the cases in which rubber tube inserts between the cut ends of the ducts have been employed intermittent attacks of chills and jaundice, indicating infection, have occurred, or else progressively deepening jaundice, indicating contraction and stricture formation. The distinct possibility of an unfortunate complication of this kind must be accepted, we believe, in all of the procedures where the sphincter of Oddi has not been preserved.

In those conditions, however, where it is impossible to demonstrate an intact distal segment of the common duct, the procedure here described, implantation into the stomach or the duodenum has no more disadvantages than the tube implantation, and, as has been already stated, it is technically much easier to employ.

It must be accepted that maintenance of patency of any scar-tissue duct is dependent upon the fact that the secretory pressure of the bile must be greater than the ability of the scar tissue in the wall of the new canal to contract. It was upon this premise that we conceived the idea of employing fistula implantation; for, as all of the external biliary fistulæ which we had seen had remained persistently open, we reasoned that the same secretory pressure which kept them open externally would maintain their patency internally.

We have now implanted three complete biliary fistulæ. The original case was implanted into the duodenum two years ago. The second was a case in which my associate, Dr. H. M.

Clute, operated upon the patient for an acute gangrenous gall-bladder, doing a cholecystectomy without injury to the duct. Following recovery from this operation the patient developed a complete biliary fistula, for which we operated twelve weeks after the original operation. An extensive carcinoma of the head of the pancreas was found, with no operative injury to the common or the hepatic duct, but there was a fistulous tract extending from the junction of the cystic duct with the common duct to the skin. An implantation of the fistula into the duodenum, by means of the technic here described, was made, with the immediate appearance of bile-colored stools and no biliary leakage. The patient, however, succumbed in a few weeks to carcinoma of the pancreas, but at no time was there biliary leakage.

The third implantation was done eleven months ago upon a patient referred to us with a complete biliary fistula, which had developed twenty months previously, immediately after the removal of her gall-bladder. The stools were completely acholic.

An implantation of the fistulous tract was made into the pyloric portion of the stomach. This part was chosen for implantation because of the immobility of the duodenum due to the dense adhesions resulting from the previous cholecystectomy, and because the mobile stomach permitted easy approximation between the fistulous tract and that organ. Following the union there was intermittent leakage of bile from the wound for twenty-one days, at the end of which time the wound closed, the stools remained well colored, and recovery was uneventful. This case, however, has twice been slightly jaundiced, with moderate chills, but is now free from jaundice or fever and is in most satisfactory condition. It is entirely possibly, however, that she may have a return of the jaundice and chills should infection again involve the bile passages by ascending along the internal fistulous biliary tract.

The technic of the procedure, as shown by the drawings (Figs. 556, 557), consists in dissection of the fistulous tract so that it is preserved intact, and the introduction of the

tract into the duodenum. A few warnings, based upon our own experience in the procedure, however, may be of value to anyone planning to employ this method.

In the dissection of the tract two precautions should be taken—one is that sufficient tissue is dissected with the tract, so that its walls may be of considerable thickness; the other is that the dissection of the tract be carried down only to the

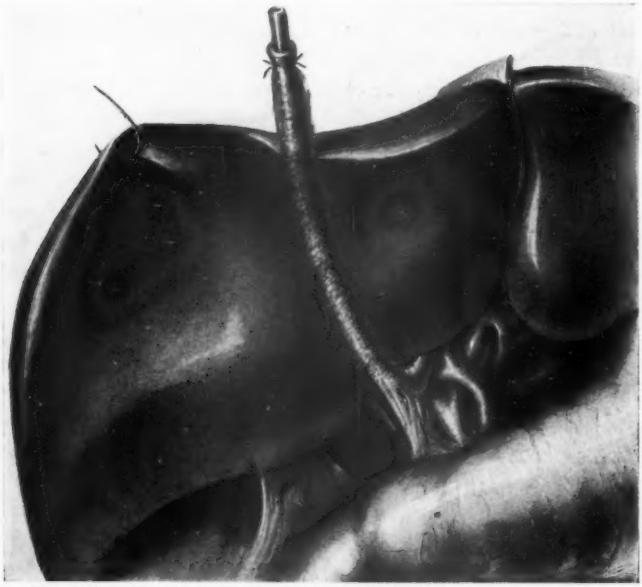

Fig. 556.—Showing fistulous tract dissected to the edge of the liver with segment of rubber tube implanted in its end.

external surface of the liver, for if the tract be separated from the under surface of the liver, which forms its inner wall, angulation and leakage is almost certain to occur. We have found that the inner portion of the sinus, the part next to the liver, is of such thinness that it does not permit freeing without leakage from the fistulous tract.

The opening in the duodenum is best made by piercing it with sharp-pointed hemostats and gently stretching the open-

ing until it just admits the end of the fistula, with a small section of rubber catheter fixed by two stitches within its open end. A purse-string suture is then inserted about the opening, and tied, not too snugly, about the implanted fistula, the rubber tube fixed in the end of the fistulous tract, preventing the collapse of the tract when this stitch is tied. Two fixing stitches may now be applied on either side to prevent pulling away of the

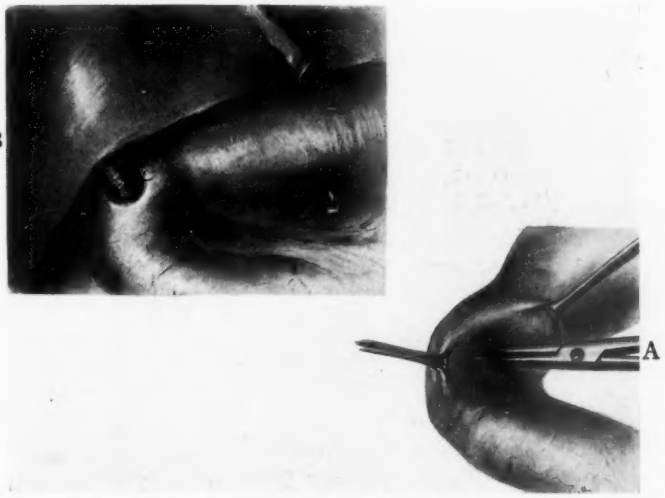

Fig. 557.—A, Showing method of making the opening into duodenum by separating with sharp-pointed hemostats to preserve muscle-fibers. B, Anastomosis complete—the fistulous tract being inverted into the bowel wall by a purse-string suture.

duodenum during the period when postoperative vomiting occurs. We have employed no drainage in these cases.

Conclusions.—In those cases particularly where the lower end of the bile duct is destroyed or is not discoverable, implantation of the biliary fistula into the duodenum offers a method of restoring bile to the intestinal tract which is exceedingly simple of execution, and possesses neither more nor fewer possibilities of postoperative complications than the methods heretofore employed.

THE TREATMENT OF DUODENAL FISTULÆ BY SUCTION

FRANK H. LAHEY

WE have recently had 2 cases of duodenal fistulæ successfully treated by suction, and we wish to present the method of applying suction which we have used in these 2 cases, together with a few remarks upon the subject.

The first case was in no way remarkable, and its details are, for the most part, unnecessary. The patient gave a history of pain in the right upper quadrant, of rather rapid onset, followed by quite acute tenderness in the right hypochondrium. He was operated before the students' clinic and found to have a perforated duodenal ulcer, around which walling off had taken place, with production of a small subhepatic abscess. As a result of drainage of this abscess a duodenal fistula developed. Following treatment by the method here described the irritating discharge promptly ceased and the wound healed rapidly.

The second case presents a point of interest other than the successful management of a presumed duodenal fistula because of the number of days following the operation before the fistula occurred. We removed a calloused pyloric ulcer by a pylorectomy, done with an anticolic anastomosis by the Polya method, on October 23, 1923, a very satisfactory recovery being made. On January 19, 1924, nearly three months later, the patient developed a tender spot in his wound, which, when opened, discharged a thin watery material that soon digested the surrounding skin and converted the sinus, which at first was most minute, into one which was rapidly and progressively enlarging. The patient returned to the hospital and after twenty-one days of suction the sinus was closed.

The suction apparatus employed in such cases is the ordinary faucet attachment suction apparatus, as shown in Fig. 558.

This apparatus has the advantage, first, of being exceedingly inexpensive; second, of being noiseless, which is a decided im-

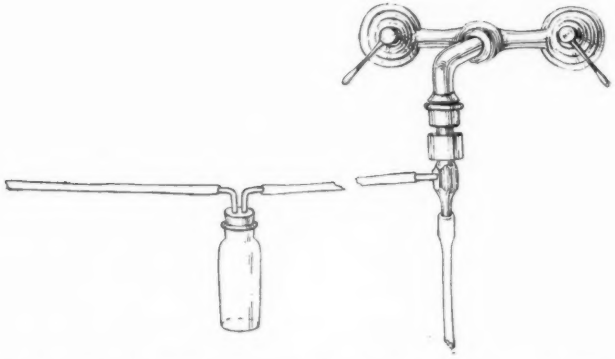

Fig. 558.—Suction apparatus attached to bath-room fixture. Suction bottle.

provement over the electric motor; and, third, that very fine adjustments in the degree of suction can be obtained by regulating the amount of water which the faucet permits to escape.

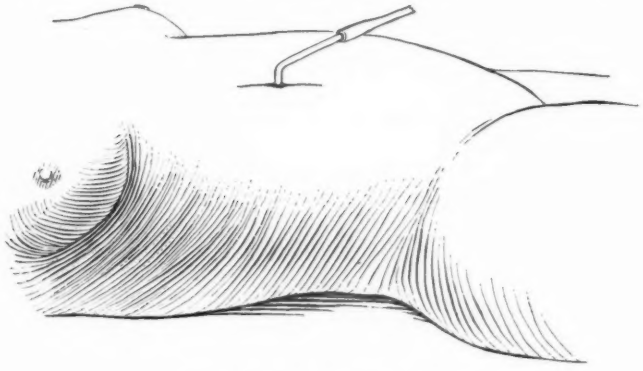

Fig. 559.—Suction tube in wound.

In the beginning a small catheter is inserted into the wound, and a flow of water permitted to escape through the suction

valve to sufficiently extract all of the fluid discharge from the wound (Fig. 559). As the wound diminishes in caliber the size of the catheter is diminished until the sinus becomes too small to admit even a baby catheter. At this time a No. 16 catheter is laid on the belly wall, as shown in Fig. 560, so that the eye of the catheter is directly over the opening of the sinus, and the suction so adjusted that any escaping fluid is promptly

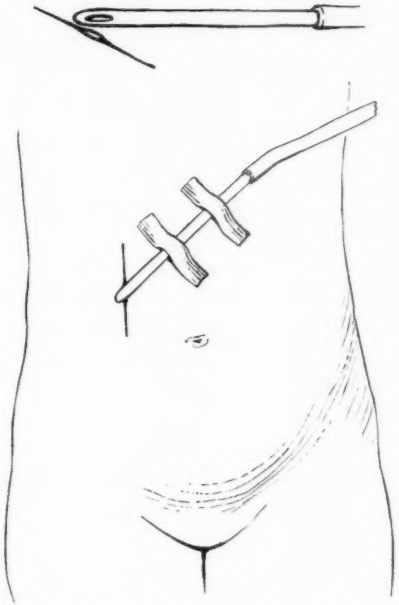

Fig. 560.—Method of applying eye of catheter over sinus after it has become too small to take a catheter.

caught up by the intaken air and deposited in an 8-ounce bottle, as shown in Fig. 558. This constant intake of air produces a slight hissing sound which is of decided value, as, when it ceases, the patient is at once aware that the eye of the catheter is blocked (usually by a small bit of slough) and requires cleaning. He may verify his suspicion that the catheter is blocked by observing the suction tube, which, being of soft rubber, will

be found to be collapsed from negative pressure when the eye of the catheter is occluded. If the sinus has been draining, he may likewise observe in the drainage bottle that no drip occurs from the tube leading from the sinus. The patient is also able to tell when the discharge from the sinus is blocked by the reaction immediately resulting from its contact with the irritated skin.

The suction valve may be attached to a faucet in the patient's room or to one in the toilet or bathroom adjacent to the patient's room, any length of tubing required to reach the patient's bed being used.

As soon as the sinus has become so small that the catheter must be laid on the skin with its opening over the wound, the patient may sit up in a chair during the day, since the discharge is as efficiently cared for in this position as in the reclining position in bed.

The skin about the wound may be protected by zinc oxid ointment with stearate of zinc dusted over the ointment.

Another point, which did not require attention in the 2 cases here cited, but must, nevertheless, be remembered, is that in cases where considerable fluid is being withdrawn by the suction apparatus it may become necessary to supply fluids and nourishment to replace the fluid removed. This is best accomplished by means of a jejunostomy, the tube being inserted by the Witzel method and led out to the abdominal wall through a small opening in the omentum, as suggested by Dr. C. H. Mayo, in order to facilitate closure when the tube is withdrawn.

It is essential that suction be instituted immediately after duodenal fistula appears, that the caliber of the tube be sufficient to efficiently remove the discharge, that the degree of suction be properly adjusted, that the tube be not permitted to occlude, and that if a large quantity of fluid be withdrawn, it be replaced by administering food and fluids through a jejunostomy, so made that it will close itself when the jejunostomy tube is removed.

TETANY

ROBERT L. MASON

WEISS[1] in 1883 reported on 13 cases of postoperative tetany which he had collected: 8 from the clinic of Billroth, in Vienna; 2 from Albert, in Vienna; 2 from Schonborn, in Königsberg, and one from Nicoladoni, in Innsbrück. These cases had occurred after total extirpation of the thyroid gland, and Weiss attributed the disease to a "persistent condition of irritation in the anterior horns of the gray substance of the spinal cord." This, in turn, he believed due to an excitation of the vascular centers of the cervical spinal cord and medulla brought about by the ligation of the many blood-vessels encountered in thyroidectomy. The parathyroids, discovered three years previously by Sandstrom, the Swedish chemist, were not thought of as having any part in the pathogenesis of the disorder. The symptom complex known as tetany (from its resemblance to tetanus), however, had been described by the British observer, Clarke, as early as 1815.[2]

The report of Weiss is important in that it occasioned, in the succeeding years, a series of investigations, clinical and experimental, upon the relation of the thyroid and surrounding structures to tetany. The rediscovery of the parathyroids by Gley in 1890 stimulated interest, but the true relation of the glandules to tetany was not clearly shown until the work of Vassale and Generali in 1900. They applied the anatomic findings of preceding workers, and by a carefully controlled series of parathyroidectomies in the cat and dog showed that after extirpation of the parathyroid bodies there ensued a syndrome analogous to tetany in man.

There has followed a host of investigators whose work has been directed chiefly toward the pathogenesis of the disorder. In general, the investigations have centered about two points:

first, the relation of tetany to the parathyroid glandules; second, the metabolic and biochemic changes incident to the symptom complex.

Concering the first it has become universally accepted that either removal, injury, or marked interference with the blood-supply of the parathyroid bodies will be followed by an attack of tetany of severity corresponding to the damage done to the glandules.

While much that has been concluded from the second of these points of investigation is purely hypothetic, the close relationship between tetany and calcium deficiency has been definitely established. This has been brought about largely through the work of MacCallum and his co-workers. However, although a vast amount of data on the subject has accumulated, the controlling mechanism, whereby the calcium is reduced, is as yet unknown. The more recent work on the subject would show that the absolute amount of the calcium content is not as important as are the degree of its ionization and the factors regulating its dissociation. This hypothesis would explain the mechanism by which the various types of tetany are produced, and serve also to correlate the divergent findings in much of the experimental work.

Calcium salts in the blood exist in three forms[3]: (*a*) as highly dissociable compounds with protein; (*b*) as undissociated crystalloid molecules; (*c*) as free ions. The latter is, presumably, the only form in which calcium can exert its physiologic effect. The protein-bound portion represents about one-third of the total calcium. Of the remaining diffusible portion, one-half is composed of free calcium ions and the remainder of undissociated crystalloid molecules of calcium salts, thought to be present as undissociated calcium carbonate in supersaturated solution.

Rona and Takahaski,[4] from studies on the solubility of calcium carbonate, have evolved the following equation to express the relation of the calcium in the blood-stream to the concentration of bicarbonate and of hydrogen ions:

$$\frac{(Ca^{++})\,(H\,CO_3^{-})}{H^{+}} = K$$

The importance, therefore, of changes in the blood-serum, either of hydrogen ion or bicarbonate concentration, becomes evident from a discussion of the several manifestations of tetany.

There are, in addition to the tetany following postoperative hypoparathyroidism, several entities of which tetany is the conspicuous symptom. These until recently were all grouped under the heading of tetany, and all were thought to be associated with parathyroid dysfunction. Aside from the postoperative variety, however, none has been proved to show association with the parathyroid bodies.

They resemble each other in two ways: first, the increased mechanical and electric hyperexcitability of the peripheral nerves which produce the same symptom complex, and, second, a probable similarity, in its ultimate aspects, in the associated disturbed blood chemistry. It would seem, as MacCallum[5] points out, that each is a different type of tetany, differing in its genesis, but similar in that the blood, in each case, arrives at the same ultimate change, after undergoing a delicate and complicated chemical process. This ultimate change would appear to be the reduction of the ionizable calcium in the blood. As MacCallum further points out, this does not preclude the possibility that the actual effect on the nerve may be due to the relative proportion of the sodium, potassium, calcium, and magnesium ions, but even this change is really the result of the reduction in the calcium.

In cases of long-standing pyloric obstruction, with prolonged vomiting, a serious form of tetany is often manifested. The mechanism, according to MacCallum and his collaborators, is somewhat as follows:

Gastric juice is unable to pass the obstructed pylorus and is vomited. There results a continuous loss from the body of HCl. This leaves the tissues with a rapidly increasing excess of base which results in a retention of CO_2 and a rise in the bicarbonate of the blood. The alveolar CO_2 tension is increased, and the blood rendered more alkaline than normal. The symptoms of tetany intervene.

Tetany from overdoses of sodium bicarbonate arise from

a similar derangement of the normal acid-base equilibrium. The bicarbonate concentration in the blood is increased more rapidly than compensatory mechanisms can restore the normal H ion concentration. Alveolar CO_2 tension is increased and a more alkaline blood produced. It is important to note that in many of the reported cases of tetany from this source there was an associated renal insufficiency which undoubtedly played a part in the accumulation of the base in the blood.

In the same group with the above is the syndrome arising from voluntary overventilation of the lungs. An excess of carbonic acid gas is driven off from the lungs, the CO_2 content of the blood is reduced, and, in consequence, the blood becomes more alkaline. There is an immediate effort to restore the $\frac{H_2CO_3}{NaHCO_3}$ ratio upon which the hydrogen ion concentration depends. This is accomplished at first by $NaHCO_3$ passing out into the tissues. A portion of it is excreted by the kidneys. However, in the process of rebreathing, CO_2 is washed out so rapidly that there is not sufficient time for reduction of enough $NaHCO_3$ to restore the ratio. The bicarbonate concentration gains the ascendancy.

In the above group the outstanding feature is seen to be the increase in the bicarbonate concentration in the blood-serum. The *total* calcium content of the blood-serum is unchanged. However, according to the formula of Rona and Takahaski (see page 1494), it is quite conceivable that both the increase in the bicarbonate concentration and the lowering of the H ion concentration may bring about a decrease in the total amount of *ionized* calcium, and the symptoms of calcium deficiency result. According to Greenwald,[6] however, the tetany is due to the specific toxic action of the sodium ion.

Recently Salveson[7] and others have produced in the experimental animal, by the oral administration of large doses of phosphates, a tetany which is practically indistinguishable from that following extirpation of the parathyroids. This confirms the results of a number of experiments in which tetany has been produced in a similar manner. In much of the previous work the deduction was that the phosphate ion exerted a specific

toxic action. In their series, however, Salveson and his co-workers have shown, in each case, a marked reduction in the blood calcium. This, they believe, is the true cause of the symptom complex following excessive phosphate administration.

Infantile tetany is almost invariably associated with rickets. For a time it was thought that association with parathyroid dysfunction had been proved by the finding of hemorrhagic areas in the parathyroids taken at necropsy from children who had died during an attack of tetany. However, further studies have shown that the hemorrhagic areas are as common in parathyroids taken from children dying of other diseases, and again, the areas have not been found in cases where tetany was present at time of death. The marked decrease in blood calcium appears to be the outstanding feature. In one series of cases[8] this has been found to be reduced to one-half the normal quantity. The phosphorus is not increased as it is in parathyroid tetany. Bicarbonate concentration is not increased. The mechanism of the reduction in the calcium with the production of the tetany syndrome remains unexplained.

Postoperative parathyroid tetany is the only tangible state which has been proved to have definite association with parathyroid insufficiency. It may occur after operations on the thyroid gland, as a result of which there has been interference with the blood-supply of the parathyroids or in which parathyroid tissue has been removed or damaged. A marked decrease in the calcium content of the blood-serum and an increase in the phosphates are shown in the blood chemistry. The concentration of bicarbonate is not above the normal values.

In the forms of tetany in which there is no change in the bicarbonate or hydrogen ion concentration in the blood, but where the total calcium is lowered, the concentration of the diffusible portion falls with the reduction in the total calcium. When the calcium ion concentration falls to a certain level one may conclude, as in the cases where the hydrogen ion or bicarbonate concentration effect a similar reduction, that the calcium is no longer able to maintain the normal irritability of the nerves. The tetany syndrome then results.

In the search for the causative mechanism in tetany various chemical products of endogenous origin have been charged with being the specific toxic agent. Among these are ammonia, guanidin derivatives, creatin, and creatinin, etc. The theory has been advocated that these substances, due to the absence of the parathyroids as detoxicating agents, are allowed to accumulate and exert their toxic action. Swingle and Nicholas,[9] in a series of animal experiments, have recently investigated this phase of the tetany problem. While after the administration of these and other toxic substances by various methods there ensued, in some cases, muscle tremors and other abnormal neuromuscular phenomena, in none was the true tetany syndrome observed. There was no reduction in the blood calcium, nor was the condition they observed relieved by calcium. Other workers have investigated the claims filed against the guanidin derivatives as the toxic agent in tetany, and, as a whole, the results throw considerable doubt on the conception that these substances play an important part in the disorder.

The symptoms and signs of tetany are brought about by the hyperexcitability of the peripheral and central nervous system. In the complete and severe form there are convulsions, spasmodic contractures with the hands in the characteristic accoucheur's position, rapid respiration, salivation, and death in coma. To the mild or transitory type belong the cases following overdoses of sodium bicarbonate, phosphate administration in large doses, voluntary overventilation of the lungs, pyloric obstruction, and in cases following thyroidectomy where there has been either partial parathyroidectomy or interference with the blood-supply of the glandules. The tetany following pyloric obstruction may be of the severe type. Infantile tetany may be of either type.

In the recurring and less severe type it is possible to observe the development of the symptoms with greater deliberation. In this clinic the earliest complaints of the four transitory types which we have observed have been stiffness and tingling in the hands and feet, with the fingers in the characteristic position of the accoucheur's hand. The fingers were flexed at the meta-

carpophalangeal joint and the head flexed at the wrist. The thumb was adducted and drawn downward toward the palm; the arms flexed at the elbow. A photograph of the hands in a recent case is shown (Figs. 561, 562). Observers in another clinic[10] have noted a twinge in the muscles supplied by the facial nerve as a first and premonitory symptom. In one of our cases a numbness of the tongue was the first symptom. Other characteristic signs of the condition are Trousseau's sign, a contraction particularly of the fingers and thumb on pressure over the large nerves of the arm. This is best elicited by applying the cuff of a blood-pressure apparatus and raising the pressure to about 200

Fig. 561.—Lateral view of accoucheur's hand, characteristic of tetany. Case recently seen in this clinic.

mm. of mercury. As pointed out by Barker,[11] the test should not be considered negative until pressure has been exerted for five minutes. In a recent case, the hands of which are shown in the photograph, all symptoms had disappeared within an hour after they were first noticed. However, three hours later the accoucheur's hand phenomenon was produced by four minutes' pressure with the sphygmomanometer cuff. Chvostek's sign may be elicited by tapping over the trunk of the facial nerve, the pes anserinus, just in front of the lobe of the ear. The resultant spasm may affect all the muscles of the face supplied by the facial nerve or single divisions. In the one case previously mentioned the spasm affected only the ala of

the nose. This, however, was definite. Pool's sign, a contracture of the muscles of the forearm, is elicited by forcible abduction of the arm. Contractures of the muscles of the lower leg may also be seen, as described by Pool,[12] by forcible flexion of the thigh or extension of the knee. In one case recently studied this sign was shown only in a plantar flexion of the foot when the knee was forcibly extended. A very constant and graphic sign, although not readily applicable in most surgical clinics, is Erb's sign, an increased irritability of the peripheral nerves to weak galvanic currents.

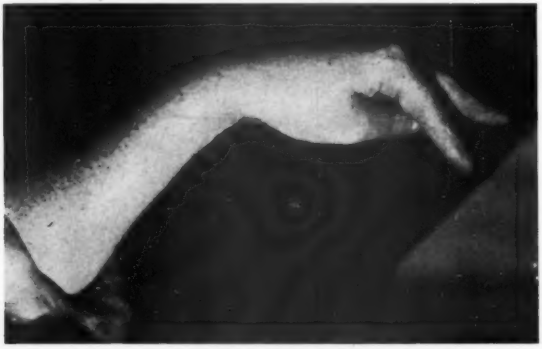

Fig. 562.—Medial view of hand shown in Fig. 561.

The normal blood calcium of 10 milligrams per 100 c.c. is reduced to 6 or 7 mg. The CO_2 combining power is not altered. The blood phosphorus is usually increased.

In chronic tetany trophic changes may occur. There may be changes observed in the nails, hair, and teeth. Many writers have reported the development of cataract in persons suffering from chronic tetany.

The treatment of tetany depends, to a certain extent, upon the type. In those varieties due to pyloric obstruction MacCallum and his co-workers have obtained relief by large doses of sodium chlorid. This they introduced in some instances by the duodenal tube; in others, intravenously. The rationale of this treatment is, of course, to replace the chlorin ions lost

by vomiting. Tetany from overdoses of sodium bicarbonate may be treated by the administration of acids, in dilute form, by mouth or intravenously, in order that the proper acid-base ratio may be restored.

In infantile tetany the restoration of the blood calcium to its normal level results, usually, in prompt disappearance of the symptoms. One should make sure, especially in adult post-operative tetany, that the condition is one of true tetany before instituting treatment. Hysteroid or tetanoid symptoms, extremely transient in nature, often manifest themselves after operation.

By mouth calcium is best given in the form of calcium lactate, since this preparation is less irritating to the stomach than calcium chlorid. Since a large portion of the calcium, when it reaches the intestine, is taken up to form soaps and excreted, leaving only a small portion to be absorbed, large doses should be given. In the adult 60 grains may be given every four hours until the symptoms are relieved. The calcium lactate should be dissolved in water before administration. In the infant 15 grains of calcium lactate may be given every four hours.

By the intravenous route calcium chlorid, 5 c.c. of a 10 per cent. solution, may be given. Care should be taken not to inject any of the drug in the tissues around the vein, since sloughing may occur. Following the intravenous administration of the calcium chlorid the patient usually complains of a sensation of warmth. This passes off within a few minutes. Nausea sometimes occurs. The drug should not be given intravenously more than once daily. It has been held by some observers that calcium by vein has a deleterious effect on the kidneys.

In cases where the contractures are severe and accompanied by great pain, relief may be secured by the subcutaneous injection of a 25 per cent. solution of magnesium sulphate, 1 c.c. for 2 pounds of body weight.

Unfortunately, in the treatment of hypoparathyroidism, the feeding of the deficient substance, parathyroid gland, has not proved successful. Dr. F. H. Lahey a few years ago, in an

effort to ascertain the value of parathyroid tetany in the various clinics throughout the country, sent out questionnaires asking the type of treatment most successful in combating tetany. Of 17 replies only one reported successful control by parathyroid administration alone. The patient so treated, however, died in epileptic convulsions some time later. Seven reported successful control by calcium treatment. Two used a combined treatment of calcium and parathyroid, with good results. The remainder had used parathyroid only, with unfavorable results.

BIBLIOGRAPHY

1. Weiss, N.: Wien. Med. Wochenschr., 1883, xxiii, 683.
2. Clarke, J.: Commentaries On Some of the Most Important Diseases of Children, London, 1815. (Quoted by Barker.)
3. Howland, J., and Kramer, B.: Monatschrift f. Kinderheilkunde, 1923, xxv, 279.
4. Rona, B., and Takahaski, D.: Biochem. Zeitschr., 1913, xlix, 370.
5. MacCallum, W. G.: Medicine, 1924, ii, 137.
6. Greenwald, I.: Jour. Biol. Chem., 1922, liv, 285.
7. Salveson, H. A., Hastings, A. B., and McIntosh, J. F.: Jour. Biol Chem., 1924, lx, 311.
8. Kramer, Tisdale, and Howland: Amer. Jour. Dis. Child., 1921, xxii, 431.
9. Swingle and Nicholas: Amer. Jour. Physiol., 1924, lxix, 455.
10. Mayo Clinic: Boothby (Discussion), Endocrinology, 1924, viii, 662.
11. Barker: Endocrinology and Metab., vol. i.
12. Pool, E. H.: Annals of Surgery, 1907, xlvi, 507.

FUNCTIONAL DISEASE OF THE COLON, DIFFERENTIATED FROM APPENDICITIS AND CHOLECYSTITIS

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ONE is frequently confronted with the problem of deciding whether a case referred as acute or recurrent appendicitis or cholecystitis is, in fact, suffering from inflammation of the gall-bladder or appendix, or is suffering cramp-like abdominal pain and tenderness as a result of irritation of the large bowel. Every surgeon has a series of cases in which he has explored the abdomen with the hope of finding an inflamed or infected gall-bladder or appendix to explain abdominal pain and tenderness only to find innocence in both these areas. In such cases the postoperative histories show usually freedom from pain for a period of several weeks to several months, but a recurrence of the same symptoms thereafter and a disillusioned patient.

That irritation and malfunction of the colon can, and frequently does, simulate both gall-bladder disease and appendicitis has been proved to the satisfaction of this clinic in numerous cases both by exploration with negative results and subsequent effective treatment for the true condition, and by "antioperative" diagnosis and bowel management, with adequate results. It therefore seems pertinent to review briefly the symptoms and signs of each of these three conditions and their possible differentiation.

The history is here as elsewhere of great value. The association of the onset of pain with cold-taking, the excessive use of irritating foods, or with so-called constipation and the abuse of cathartics is very frequent in cases of bowel distress and very rare in appendicitis and gall-bladder disease. "Constipation," especially, with its secondary meaning of the use of all kinds and large quantities of cathartics, usually has a definite causal

relationship to bowel distress and a much less definite and constant relationship to appendical and gall-bladder disease.

In colon irritation there is usually a history of pain which shifts in location, but which at some period in the history extends below the umbilicus and in both lower quadrants. Though the patient usually ascribes it to either the right or left side, he is unable to localize definitely a single area of maximum pain. Appendicitis pain, though frequently shifting at the onset, is usually well localized after a few hours to the area over the appendix which ordinarily varies within small limits. Pain from gall-bladder disease is characteristically well localized under the right subcostal area, and radiation downward below the umbilicus is rarely noted. Radiation to the right dorsal area, characteristic of gall-bladder pain, is not seen in colonic pain, though the latter does occasionally radiate to the sacroiliac region as does appendical pain occasionally. The character of the pain in all three conditions may be the same, lancinating and cramp-like, though the pain of bowel distress varies greatly from a mild, shifting, come-and-go pain to colic. Bowel distress frequently occurs directly after food taking, but this may also be true of appendix and gall-bladder pain.

Tenderness is present in appendicitis and gall-bladder disease and usually likewise in irritable bowel. In cholecystitis it is definitely localized in the right upper quadrant. In appendicitis it is present over the inflamed appendix, the location of which may vary in the right side of the abdomen, but is usually below the level of the umbilicus. Left-sided appendical tenderness is sufficiently rare to cause little difficulty in differentiation. Colonic tenderness is characteristically present over the entire course of the colon, but a confusing situation arises when, as occasionally happens, it is likewise limited to a small area in the cecum, ascending colon, or hepatic flexure. Palpation of the colon usually discloses a spastic condition which makes it possible to roll the colon under the fingers on either side of the abdomen. Spasm of the abdominal wall is usually present in appendicitis and gall-bladder disease, but may sometimes be encountered also in bowel distress.

Vomiting is commonly associated with appendix or gall-bladder pathology and does not occur with uncomplicated colon pathology. Nausea, however, is occasionally present in the latter condition, and when present is usually associated with dizziness and gaseous eructations. The two last named symptoms may be the sole manifestation of colonic malfunction.

Loss of appetite, strength, and weight are frequently associated with all three conditions and are of no value in differentiation except in the fact that these, like the other symptoms, are likely to have been continuous over a long period of time in bowel distress. The date of onset of the condition is not of all-important significance, however, as the patient may present himself for examination immediately after the onset of bowel distress, which is severe, as well as after the onset of acute pain of gall-bladder or appendical origin. An intermittent history is more significant for appendicitis or gall-duct obstruction than for bowel distress unless the history of irritating cause in the latter condition is concomitant.

Urinalysis is of no aid except where the presence of bile indicates a disturbance of biliary drainage. An elevated temperature and white count are, of course, indicative of inflammatory disturbance and indicate something more than a simple functional disturbance of the colon. The icterus index should theoretically aid in locating the cause of the disturbance in the right upper quadrant, but in the experience of this clinic up to the present it has not been of value. Blood examination is in other respects of no value in differential diagnosis.

Analysis of the gastric contents after an Ewald meal usually shows in uncomplicated appendicitis a normal free HCl and total acidity, whereas gall-bladder pathology is frequently accompanied by an achlorhydria. In bowel distress due to the usual causes the free and combined acidity are likewise normal, but there does exist a type of bowel distress and diarrhea due to an achlorhydria and relieved by the administration of dilute hydrochloric acid.

In none of these conditions is the stool characteristic except that the stool of gall-bladder disease may fail to show normal

bile-pigments and the stool of an irritable colon with diarrhea may show undigested food or too great or too little dehydration according to the phase of bowel irritation. Mucus may also be present in the latter condition.

x-Ray is of considerable value in the differentiation of these conditions. The most reliable aid afforded by *x*-ray in the diagnosis of appendicitis is the visualized appendix tender to palpation under fluoroscopy. *x*-Ray diagnosis of gall-bladder pathology is simple when there are visualized calculi. Cholecystitis without visualized stones is, however, elusive to *x*-ray even with the newer methods of dye injection. Cases have been explored in this clinic where a preoperative diagnosis of gall-bladder disease was made on the fact that the gall-bladder failed to fill normally with dye and no pathology was found. In a few cases the normal filling of the gall-bladder was substantiated by operative findings.

The irritable bowel, on the other hand, is demonstrated with a moderate degree of constancy by the barium enema which fills the colon with greater smoothness and speed than normally. Emptying is likewise more rapid than normal, and after emptying the descending portion especially hangs like a string. The characteristic pain of which the patient has complained is reproduced during the injection, and the filled colon may be palpated under the fluoroscope and found to be tender along its course.

A test used in the Sippy Clinic and of value in differentiation is the test-out enema, a 6-pint enema of water at body temperature, given 1 pint at a time. The onset and character of distress which may occur during the injection is recorded and its character compared with that of which the patient originally complained. If they are identical, the original distress was probably that of irritable bowel.

The relief measures for the three conditions under discussion are usually different and serve to distinguish them. While the pain of appendicitis may be relieved at least to some extent by cold and that of cholecystitis usually requires morphin or codein, bowel distress may be relieved usually by the external

application of heat or the drinking of hot water, by a bowel movement, or frequently by the belching of gas or passing of flatus. It is increased in intensity when the patient moves about or takes cold.

The association of irritable colon with both appendicitis and gall-bladder disease is not unusual, and in such cases the picture is confused. A case recently seen in the clinic was admitted because of severe right-sided cramp-like pain, for which her family physician wished immediate exploration. One month before her admission the patient's husband had died of general peritonitis due to ruptured appendix, and this fact produced in both family physician and patient great apprehension. She gave a definite history of cause for bowel irritation, namely, the continued use of magnesium sulphate for the purpose of reduction in weight, with the onset of abdominal pain a week after the beginning of this treatment. The pain had increased in severity during the week before admission, but remained of the same character until in the forty-eight hours preceding admission it became unusually severe and, in addition to the shifting pain and tenderness, there was increasingly severe pain in the right lower quadrant. There was no elevation of temperature or white count, her abdomen was slightly more spastic in the right lower quadrant than in the left, and there was definitely more tenderness in the area of the cecum than in that of the rest of the colon. Rectal examination was negative. Although it was felt that even her extreme tenderness over the appendical area might possibly be due to irritable bowel, it was decided by Dr. Lahey that exploratory laparotomy should be done. A definitely acute appendix with fibrin was found. It was interesting to note that appendectomy did not relieve the shifting pain which had been present before the onset of the more severe localized pain. Bowel management was begun a week after operation with subsequent complete relief of all pain, and several months later the patient reported normal bowel function and no recurrence of symptoms.

Conclusions.—It is important to distinguish between this condition of bowel distress, entirely amenable to medical treat-

ment, and the two surgical conditions with which it is frequently confused. A careful study of the signs and symptoms discussed above usually results in the prevention of surgery in cases of bowel distress, but where there is cause for a reasonable doubt, it remains the part of wisdom and good judgment to explore first and to use medical management subsequently.

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